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DONALD L. McRAE
Caldwell Lecturer, 1959

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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 84 JULY, 1960 NO. 1

THE SIGNIFICANCE OF ABNORMALITIES OF THE CERVICAL SPINE*

CALDWELL LECTURE, 1959

By DONALD L. McRAE, M.D.
MONTREAL, QUEBEC, CANADA

IT IS an honor to be invited to give the Caldwell Lecture to the American Roentgen Ray Society. I did not know Dr. Caldwell but was aware that he was a pioneer American radiologist. For many years, I have used Caldwell's projection as a necessary part of a complete examination of the skull (Fig. 1). It shows the floor of the anterior fossa (the orbital roof), the anterior walls of the middle fossa (the greater wings of the sphenoid bone), the superior orbital fissures and the frontal and ethmoid sinuses as well, if not better, than any other single projection. I am indebted to many of the previous Caldwell lecturers, particularly to Dr. Hickey for his lecture of 1928, for a better understanding of this important figure in American radiology. Every student of radiology could profit from thoughtful reading of Dr. Hickey's description of Caldwell's life.

In 1904, in the preface to his book "Practical Applications of X-ray in Diagnosis and Therapy," Caldwell stated that there was a tendency to either underestimate the value of the roentgen ray in diagnosis or to fail to appreciate its limitations. That this is still true is shown by occasional articles and editorials in nonradiologic journals decrying the value of radiology in diagnosis. Such articles reveal a rather widespread lack of understanding of the actual value of roentgenologic methods of examination. The roentgenologic examination reveals anatomic, pathologic and physiologic facts that are incontrovertible but does not reveal their significance in relation to the patient's signs or symptoms. In ordinary words, we can say that the roentgenologic examination shows what is there but does not tell whether symptoms are resulting from it. I believe that radiologists should go beyond morphologic and physiologic interpretations whenever possible and attempt to assess the significance of their findings. Of course, this is best done in personal consultation with the patient's physician.

The cervical spine is an area in which roentgenologic examination frequently shows pathologic changes in asymptomatic as well as in symptomatic persons. It is a frequent site of pain and stiffness. The cervical spinal cord is often involved in

* Presented at the Sixtieth Annual Meeting of the American Roentgen Ray Society, Cincinnati, Ohio, September 22–25, 1959.

Fig. 1. Posteroanterior roentgenogram of the skull in Caldwell projection.

neurologic diseases such as multiple sclerosis, syringomyelia, amyotrophic lateral sclerosis, subacute combined degeneration and so on. Therefore, radiologists frequently have patients who have symptoms and signs that may be due to cervical spine or cervical cord disease and whose cervical roentgenograms are abnormal. In such cases, the pathologic changes revealed on roentgenograms must correspond with the level of symptoms and signs before the roentgenologic changes can be considered of probable etiologic significance. Unfortunately, neurologic localization in the upper cervical cord is not precise and, besides this, spinal cord lesions may not be confined to one segment of the cord. Therefore, we must seek for roentgenologic criteria that will help in deciding whether or not the pathologic changes revealed on the roentgenograms are, or are not, significant.

Bony abnormalities about the foramen magnum frequently produce neurologic signs and symptoms which point to various levels in the central nervous system.

Table I shows the most common complaints in a group of 68 patients taken from the records of the Montreal Neurological Institute up to the end of 1955, with symptom-producing bony abnormalities about the foramen magnum. Uncommon complaints were dizzy spells, spells of ringing in the ears, episodes of visual blurring, episodes of diplopia and weakness of one arm. In these patients, a history of exacerbations and remissions was often obtained. Different diagnoses were suggested by different physicians who saw the patients at different stages in their disease. The referring diagnoses in the 68 patients are shown in Table II.

Many of the conditions listed in Table II are those for which no curative treatment exists at the present time. Bony abnormal-

Table I

BONY ABNORMALITIES ABOUT THE FORAMEN MAGNUM

Chief Complaint	Frequency (per cent)	Average Duration	Average Age at Onset
Weakness of one or both legs	32	7 yr. (1–20 yr.)	31 yr. (12–47 yr.)
Occipital or suboccipital pain	26	3 yr. (1 mo.–9 yr.)	28 yr. (7–51 yr.)
Neck pain or paresthesias	13	5 yr. (6 mo.–17 yr.)	30 yr. (16–40 yr.)
Numbness or tingling of fingers	12	2 yr. (1–4 yr.)	43 yr. (21–57 yr.)
Ataxic gait	9	18 yr. (1–49 yr.)	33 yr. (5–50 yr.)

TABLE II

BONY ABNORMALITIES ABOUT THE FORAMEN MAGNUM

Diagnosis of Referring Physician	
Multiple Sclerosis	21
Syringomyelia or Syringobulbia	12
Brain Stem Tumor or Posterior Fossa Tumor	11
Foramen Magnum Lesion or Arnold-Chiari Malformation	9
Cervical Fracture or Dislocation or Cervical Disk Protrusion	6
Degenerative Disease of Cord	4
Cerebellar Degeneration	3
Hysteria	2
Lead Poisoning (chronic)	1

ities about the foramen magnum, on the contrary, are amenable to surgical treatment, which in most cases either arrests the progress of the disease or leads to considerable improvement. Bony abnormalities about the foramen magnum are not uncommon.

CHRONIC ATLANTO-AXIAL DISLOCATION

In 1953, in a report on bony anomalies at the craniovertebral junction, I described 6 cases of chronic atlanto-axial dislocation.[3] I used the word "chronic" because in none of them was there a history of recent head or neck injury and in all of them there was good reason to believe that the dislocation had been present for a long time. The fact that none of them gave a history of old neck injury made it probable that some, if not all, of the lesions were congenital rather than acquired. We have had no more cases since that time. These were pure cases, *i.e.*, there was no other significant primary bony abnormality in this region. In most of them, the atlas was dislocated directly forward (Fig. 2, *A* and *B*). The odontoid process of the atlas in such cases presses on the lower part of the medulla oblongata and the upper spinal cord. Neurologic symptoms and signs are present with sagittal dislocations of a few millimeters. Marked sagittal dislocation at this level is incompatible with life. If, however, the atlas dislocates to one side as well as anteriorly, the medulla and spinal cord may slip around the odontoid process and escape compression (Fig. 3, *A* and *B*), but this occurred only once in our series. Two patients showed slight lateral displacement in addition to the sagittal displacement (Fig. 4, *A* and *B*).

Four of these patients had neurologic signs and/or symptoms which we felt were due to chronic compression of the lower medulla oblongata and upper spinal cord. The neuraxis was squeezed between the odontoid process of the axis and the posterior arch of the atlas as shown in the diagram from Watson-Jones[8] (Fig. 5, *A* and *B*). The diagram also shows why greater dislocation can be present without neurol-

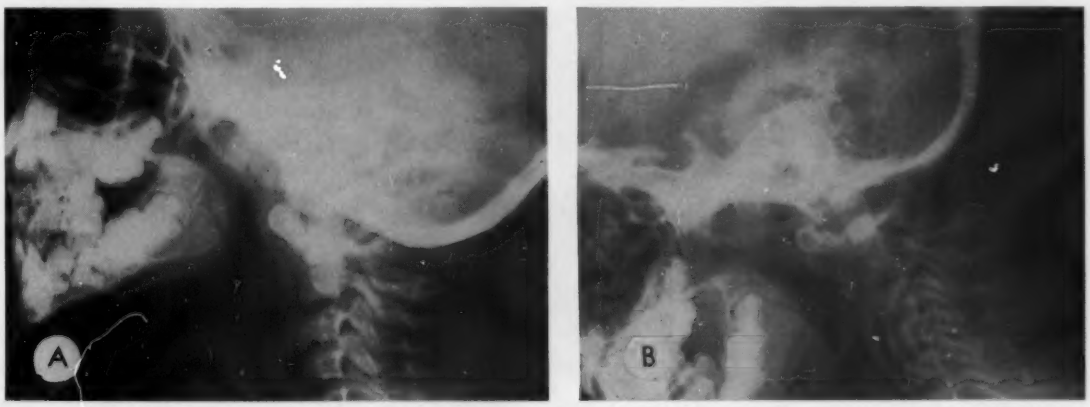

FIG. 2. (*A* and *B*) Anterior atlanto-axial dislocation in a boy aged five. He was mentally retarded and had torticollis as well as a club foot but no localized abnormal neurologic signs.

TABLE III

CHRONIC ATLANTO-AXIAL DISLOCATION

6 Cases: 3 male, 3 female

Neurologic Symptoms in 4 Cases		Neurologic Signs in 3 Cases	
Weakness or ataxia—arms	2	Long tract deficit—arms	1
Weakness or ataxia—legs	2	Long tract deficit—legs	3
Numbness or pain—arms	3	Ataxia—arms	1
Numbness or pain—legs	1	Ataxia—legs	0
Headache	1	Nystagmus	0
Neck pain	3	Bulbar signs	0
Visual symptoms	0	Posterior column deficit—arms	0
Auditory symptoms	0	Posterior column deficit—legs	0
		Two point loss astereognosis—arms	1
		Two point loss astereognosis—legs	0

ogic symptoms in cases of a separate odontoid process, which is to be described later. None of the cases of pure chronic atlanto-axial dislocation has come to autopsy so a complete morphologic description cannot be given. The 4 symptomatic patients were operated upon. In none was there a visible abnormality of the central nervous system such as an Arnold-Chiari malformation which might have produced some or all of the symptoms. In Table III the symptoms and signs which were noted in these cases of chronic atlanto-axial dislocation are listed.

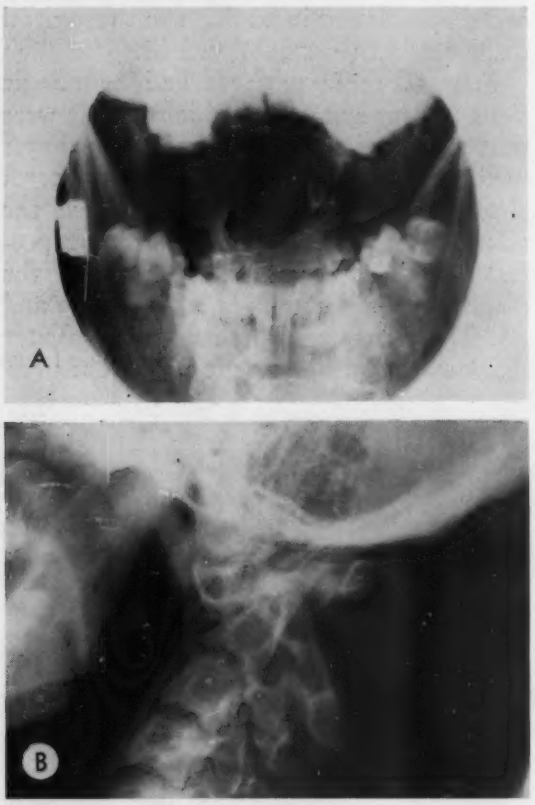

FIG. 3. (*A* and *B*) Oblique atlanto-axial dislocation in a girl aged sixteen. She was mentally retarded but there were no other abnormal neurologic signs.

When such cases are seen, they are apt to be diagnosed as rupture or aplasia of the transverse ligament of the atlas. Such a lesion may be present but never as the sole lesion. There are many other ligaments holding the atlas to the axis, notably the ligamentum nuchae and the ligamenta flava. These also must be torn, stretched or absent in order to allow the atlas to move forward on the axis. It is important that these other ligaments are considered as well as the transverse ligament of the atlas.

Chronic atlanto-axial dislocation is important in association with both simple and complex bony abnormalities of the craniovertebral junction. Lateral cervical spine roentgenograms should always be made in full flexion and full extension in patients with lesions of the cervical spinal cord or lower medulla oblongata in order that atlanto-axial dislocation can be determined. Its presence indicates potential if not actual compression of the neuraxis as List[1] emphasized in 1941.

SEPARATE ODONTOID PROCESS OF THE AXIS

Many articles have been published on the separate odontoid process of the axis but most authors have considered the lesion to be of traumatic origin. Some of the reported cases of an absent odontoid process have actually been cases of separate odontoid processes, as careful inspection of the published roentgenograms will show. At the Montreal Neurological Institute, we have not had a case of proved absence of the odontoid process; in all of our cases an odontoid process was demonstrated at least on the laminagrams. We prefer the name "separate odontoid process" to "ununited fracture of the odontoid process" because it is noncommittal and because we feel certain that some of the cases are congenital in nature.

In 1953, I reported 11 cases of this condition[3] and by the end of 1958 we have had 6 more. Symptoms and signs pointing to a lesion at the cranio-vertebral junction were present in at least half of the cases (Table IV). These are not due to the separate odontoid process itself, but to a recurrent or a chronic progressive atlanto-axial dislocation that leads to compression of the upper cervical cord. Sometimes the dislocation occurs in flexion (Fig. 6, *A* and *B*) and sometimes in extension (Fig. 7, *A* and *B*). The neurologic symptoms and signs seem to be the same in each case. Even though the central nervous system lesion is probably slightly lower than in occipitalization of the atlas and in basilar invagination, it is not possible to show this by neurologic examination.

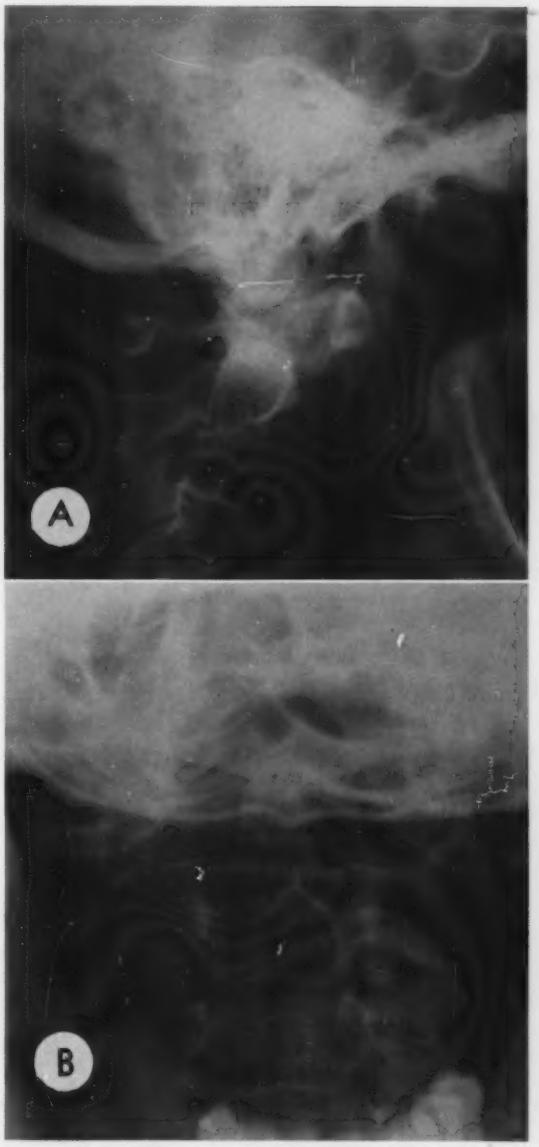

FIG. 4. (*A* and *B*) Anterior atlanto-axial dislocation in a fifty-eight year old physician whose chief complaints were numbness and tingling of the hands. He had had at least one transient quadriplegia. A diagnosis of multiple sclerosis had been made.

OCCIPITALIZATION OF THE ATLAS

In 1953, Barnum and I[2] reported 25 patients with occipitalization of the atlas and later in 1953 I added 3 more, making a total of 28. By the end of 1958, our series had reached 43. The percentage of symptomatic cases is less now than in 1953, not due to any change in the nature of the disease but to a change in the methods of selection. The first group of 25 cases was comprised mainly of patients with neurologic symptoms and signs pointing to a lesion in the general region of the cranio-vertebral junction. In this group, therefore, bias as regards the large number of symptomatic cases is evident. The subsequent 18 cases were found by carefully studying the cranio-vertebral junction on

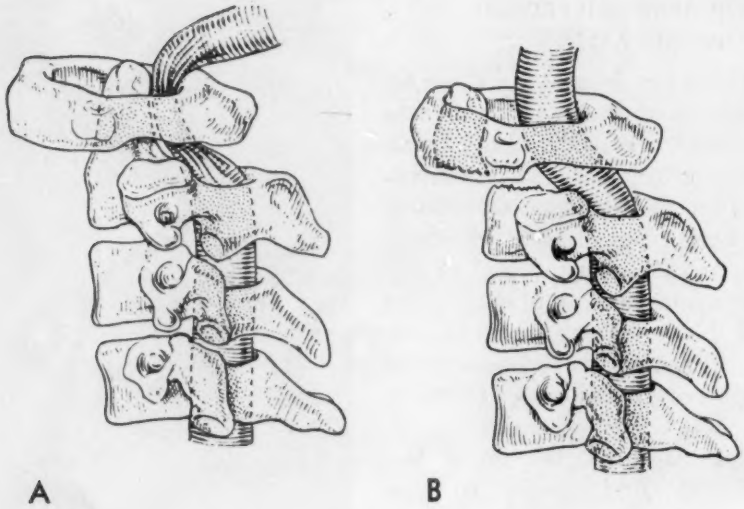

Fig. 5. (*A* and *B*) Diagram from Watson-Jones[8] showing why atlanto-axial dislocation alone is more serious than atlanto-axial dislocation with separation of the odontoid process.

all skull and cervical spine roentgenograms, whether or not symptoms referable to this region were present. Since most of these skull and cervical spine roentgenograms were taken for reasons other than symptoms suggesting lower medullary or upper spinal cord lesions, there was less likelihood of bias.

Central nervous system symptoms and signs in the patients with occipitalization, who we believe had compression of the lower medulla oblongata or the upper cervical cord, are shown in Table v.

In our first report,[2] we suggested that the symptoms were due to compression of the lower part of the medulla oblongata. The pathologic findings noted at operation or autopsy have strengthened this belief. There is a notable lack of any other congenital deformity of the bones, joints, meninges or central nervous system. The fluid collections, found inside the cervical spinal cord in several cases, we consider to be the result of softening of the cord and not evidence of primary syringomyelia. The pressure from in front is caused by the

Table IV

SEPARATE ODONTOID PROCESS OF THE AXIS

17 Cases: 12 male, 5 female

Neurologic Symptoms in 10 Cases		Neurologic Signs in 10 Cases	
Weakness or ataxia—arms	7	Long tract deficit—arms	6
Weakness or ataxia—legs	7	Long tract deficit—legs	6
Numbness or pain—arms	6	Ataxia—arms	5
Numbness or pain—legs	4	Ataxia—legs	4
Headache	4	Nystagmus	1
Neck pain	6	Bulbar signs	0
Visual symptoms	0	Posterior column deficit—arms	4
Auditory symptoms	1	Posterior column deficit—legs	5
		Two point loss astereognosis—arms	3
		Two point loss astereognosis—legs	3
		Horner's syndrome	0

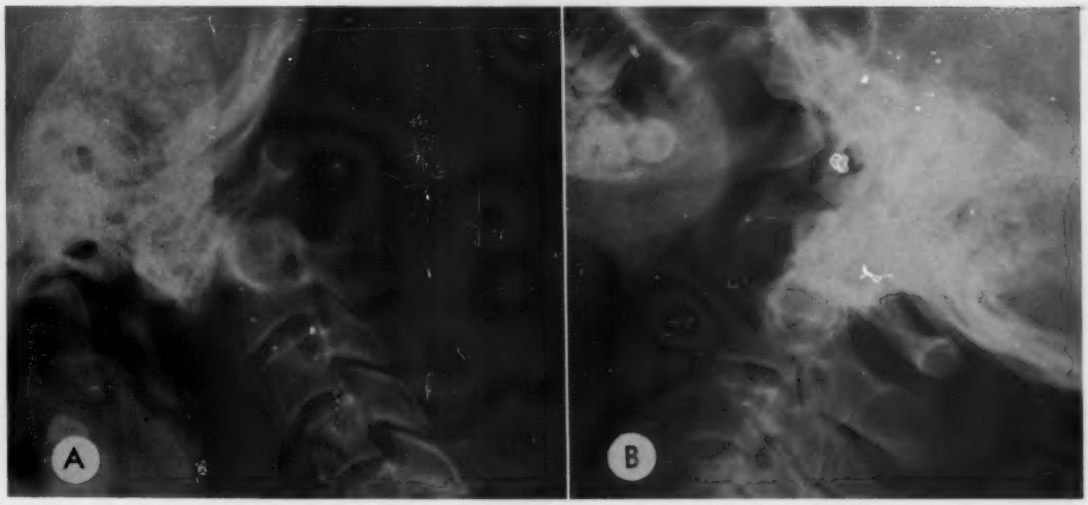

Fig. 6. (*A* and *B*) Separate odontoid process with atlanto-axial dislocation in flexion in a forty-one year old male who had marked weakness of the legs and moderate weakness of the arms.

odontoid process. Three cases at autopsy showed an impression in the ventral surface of the lower medulla oblongata produced by the odontoid process (Fig. 8). The odontoid process comes in contact with the medulla oblongata because the anterior arch of the assimilated atlas is not only high but also posterior in position (Fig. 9, *A* and *B*). As a result, the odontoid process often lies partly or wholly within the foramen magnum. Pressure from behind comes from the posterior arch of the atlas, the posterior lip of the foramen magnum or from a thick band of dura at the junction of the cranial and spinal dura. Since the posterior arch of the atlas may or may not be fused to the occipital bone and may lie anterior or posterior to the posterior lip of the foramen magnum, we try to estimate the size of the effective foramen magnum by measuring the shortest distance from the odontoid process to either the posterior arch of the atlas or the posterior lip of the foramen magnum. All of our patients showing an anteroposterior diameter of the effective foramen magnum of less than 19 mm. on at least one roentgenogram (made at six foot focal film distance) had symptoms or signs of disease in the lower medulla or upper spinal cord. In a few cases, this an-

Table V

OCCIPITALIZATION OF THE ATLAS

43 Cases: 31 male, 12 female

Neurologic Symptoms in 26 Cases		Neurologic Signs in 22 cases	
Weakness and ataxia—legs	14	Long tract deficit—arms	15
Weakness and ataxia—arms	16	Long tract deficit—legs	12
Weakness and ataxia—both	13	Ataxia—arms	8
Numbness and pain—arms	11	Ataxia—legs	7
Numbness and pain—legs	4	Nystagmus	10
Numbness and pain—both	6	Bulbar signs	7
Headache	12	Posterior column deficit—arms	6
Neck pain	8	Posterior column deficit—legs	10
Visual symptoms	7	Two point loss astereognosis—arms	7
Auditory symptoms	5	Two point loss astereognosis—legs	3

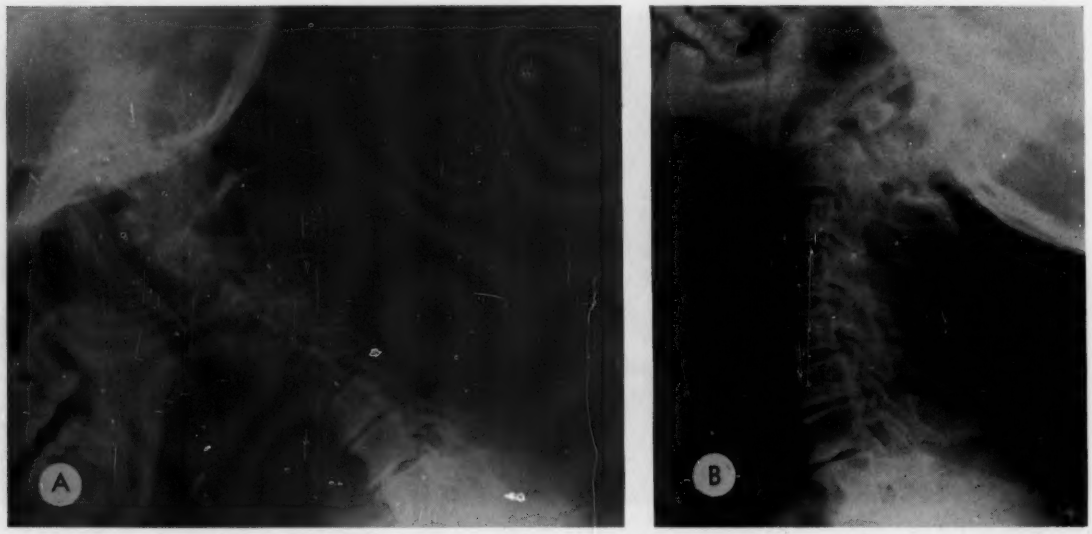

FIG. 7. (*A* and *B*) Separate odontoid process with atlanto-axial dislocation in extension in a fifty-seven year old male who had had weakness of the left foot and arm for at least eight years and recently some weakness of the right limbs.

teroposterior measurement was less than 19 mm. on the roentgenogram made with the head flexed but greater than 19 mm. on roentgenograms made with the head extended or in neutral position. This was due to partial forward dislocation of the occipitalized atlas on the axis during forward bending (Fig. 10, *A* and *B*).

At operation a dural band pressing against the posterior surface of the lower medulla was seen in 4 cases. Of course, it is not visualized on plain roentgenograms, yet it may cause pressure on the medulla oblongata when the effective foramen magnum measures more than 19 mm. In one of the 4 cases, gas myelography and posterior fossa encephalography were performed but the dural band was not visualized due to overlying bony shadows. It is possible that lateral laminagrams made

FIG. 8. Photograph of the medulla oblongata of a patient with occipitalization of the atlas showing the excavation in its anterior surface due to pressure from the odontoid process. Case F.B., male, aged twenty-two.

Fig. 9. (*A* and *B*) Occipitalization of the atlas showing the odontoid process within the foramen magnum. Case A.L., female, aged forty-six, symptomatic.

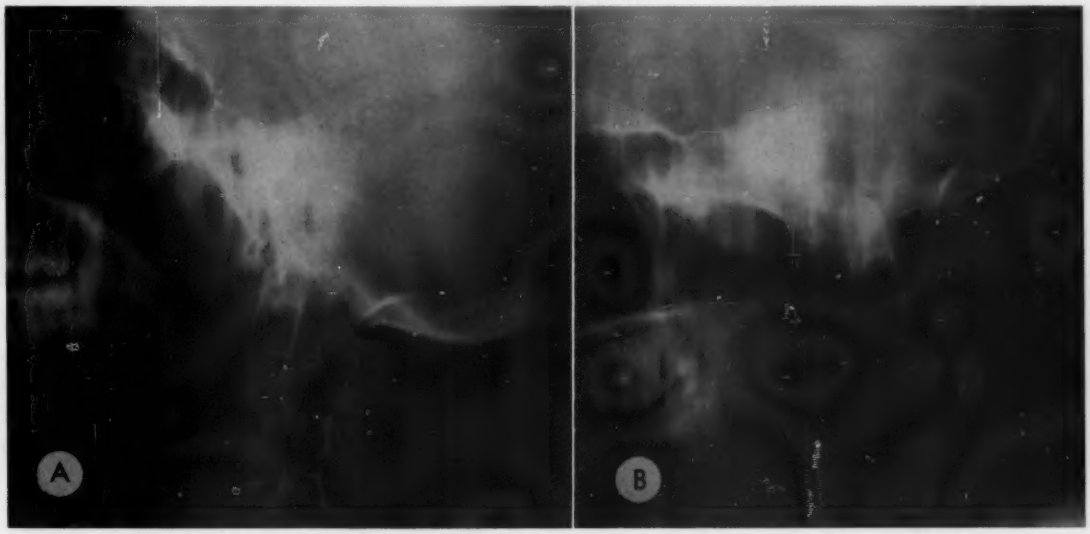

Fig. 10. (*A* and *B*) Occipitalization of the atlas. Lateral laminagrams in flexion and extension show atlanto-axial dislocation in flexion leading to constriction of the effective foramen magnum. Case A.L., male, aged twenty-two, symptomatic.

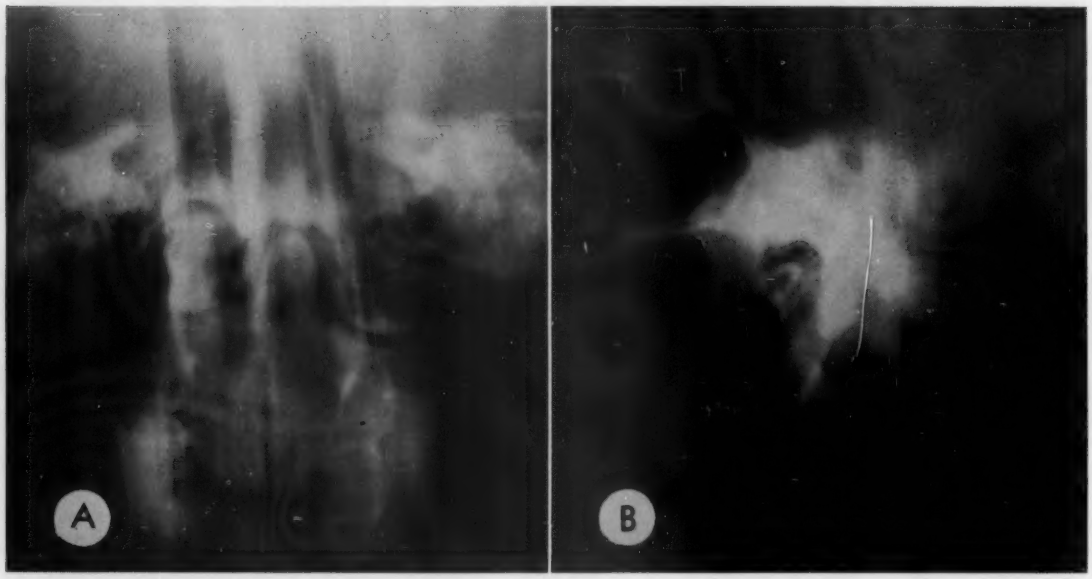

Fig. 11. Basilar invagination. (*A*) Anteroposterior laminagram showing that the lateral masses of the atlas are not fused to the skull. (*B*) Lateral laminagram showing that the anterior and posterior arches of the atlas are not fused to the skull.

during gas myelography would reveal such dural bands. I do not use oil myelography in these patients as I do not think it advisable to introduce oil into the skull.

In general, we can say that there is a congenital abnormality at the cranio-vertebral junction consisting of fusion of the atlas to the occipital bone, usually without any other associated congenital anomaly of the central nervous system or meninges. In about one-half of unselected cases of this anomaly, there were secondary atrophic changes in the medulla oblongata and spinal cord. In symptomatic cases the diagnosis can be made with fair accuracy if there is a small effective foramen magnum, if there is much atlanto-axial dislocation or if a dural band can be shown by gas myelography at the posterior lip of the foramen magnum.

BASILAR INVAGINATION

Although not an abnormality of the cervical spine, basilar invagination must be mentioned because it must be considered in the differential diagnosis of lesions at the cranio-vertebral junction and because it may be mistaken for occipitalization of the atlas. Anteroposterior laminagrams are necessary to show joint spaces between the occipital condyles and the lateral masses of the atlas (Fig. 11*A*). Lateral laminagrams of the cranio-vertebral junction are necessary to show that the anterior and posterior arches of the atlas are not fused to the occipital bone (Fig. 11*B*). I find the basal angle of little value in determining the presence or absence of basilar invagination. Most brachycephalic skulls have a basal angle of 100 to 115 degrees whether or not there is basilar invagination. Most dolichocephalic skulls have a basal angle of 130 to 140 degrees, almost platybasic, even though actual invagination is not present. Basilar invagination implies that the margins of the foramen magnum are turned upwards. Most of the lines and angles used as criteria for basilar invagination have one point or one limb away from the foramen magnum and therefore are not always reliable. In addition, the length of the basi-occiput and the slope of the posterior lip of the foramen magnum vary; therefore, the plane of the foramen magnum line is not constant. When its anterior end is high, as in Figure 12*C*, one gets the impres-

Fig. 12. Sketches of the foramen magnum as seen on lateral roentgenograms in patients with (*A*) long basi-occiput, (*B*) average basi-occiput, (*C*) short basi-occiput. Note the change in plane of the foramen magnum.

Fig. 13. Types of occipital condyles (arrows) and different planes of the atlas (a) as seen on lateral roentgenograms in patients with (*A*) average sized, horizontal, occipital condyles and a horizontal atlas in average position, (*B*) large occipital condyles producing a low position of the atlas, (*C*) oblique condyles high in position producing high position and obliquity of the atlas.

sion of basilar invagination. The prominence and slope of the occipital condyles are not constant. In a patient with large prominent occipital condyles (Fig. 13*B*), the atlas is low and so is the axis. If the occipital condyles are small (Fig. 13*C*), the atlas and axis are high and Chamberlain's and MacGregor's lines will pass through the odontoid process. When the occipital condyles and atlas lie obliquely, as seen in the lateral roentgenogram, any line or angle that makes use of the atlas will tend to be abnormal and basilar invagination may be diagnosed when not actually present. This is most frequent in patients with a short basi-occiput.

It is difficult to set roentgenologic criteria for the diagnosis of basilar invagination, especially when it is of a minor degree. Most, if not all, of the lines and angles used for this purpose will occasionally suggest invagination of the margins of the foramen magnum when such is not present.

OTHER BONY ANOMALIES AT THE CRANIO-VERTEBRAL JUNCTION

Spina bifida posterior or posterior spondyloschisis of the atlas is quite common, occurring in almost 5 per cent of all patients. By itself, it is of no importance except that it represents a weak atlas. This defect is recognized on lateral roentgenograms of the skull or cervical spine because of the absence of the shadow of the posterior wall of the spinal canal at C1 (Fig. 14, *A* and *B*). Anterior spondyloschisis of the atlas is less common (Fig. 15). It, too, represents a weak atlas but otherwise does not seem to

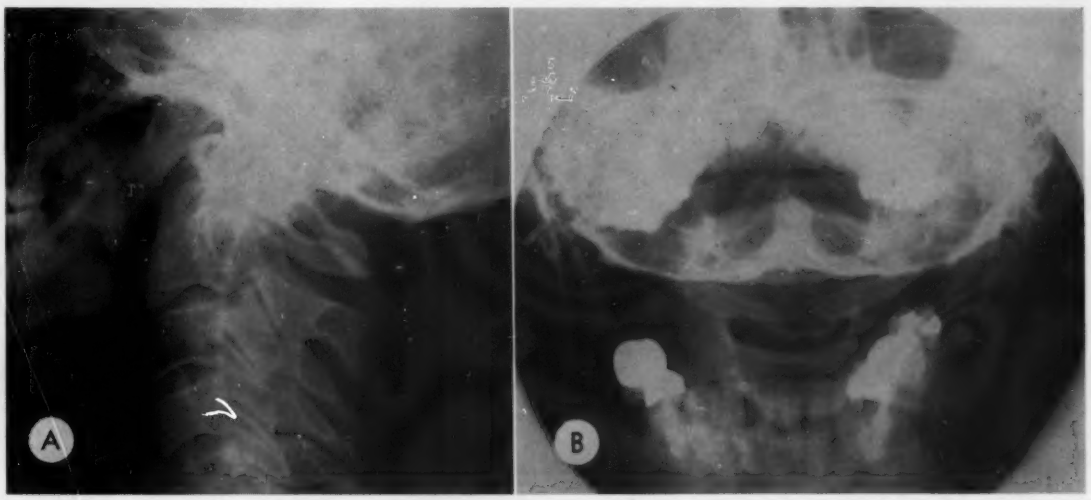

Fig. 14. (*A* and *B*) Posterior spondyloschisis of the atlas. J.S., male, aged thirty-nine, asymptomatic.

Fig. 15. Anterior spondyloschisis of the atlas. T.W., male, aged twenty-eight, asymptomatic.

Fig. 16. Absence of parts of both sides of the posterior arch of the atlas. W.C., male, aged sixty-four, asymptomatic.

be of much significance. Combined anterior and posterior spondyloschisis results in an atlas that is split sagittally. The common occurrence of this anomaly is not generally realized because roentgenograms in basal view of the skull are not made on all patients. We have had at least 6 such patients who had no symptoms or signs. In 2, and perhaps in 3, of our patients with complex upper cervical anomalies, one-half of a sagittally-split atlas was fused to the base of the skull and the other half to the axis vertebra. The bending roentgenograms of the neck did not show excessive mobility at this level in these patients.

Lateral defects of varying size in the posterior arch of the atlas are rather rare and seldom seem to be of any significance. They may be unilateral but, in general, are bilateral (Fig. 16). When unilateral, assimilation of that side of the atlas to the base of the skull may be present.

Asymmetry of the lateral facets of the atlas and axis is not uncommon and by itself is probably not significant (Fig. 17, *A* and *B*). I have never seen it in a patient

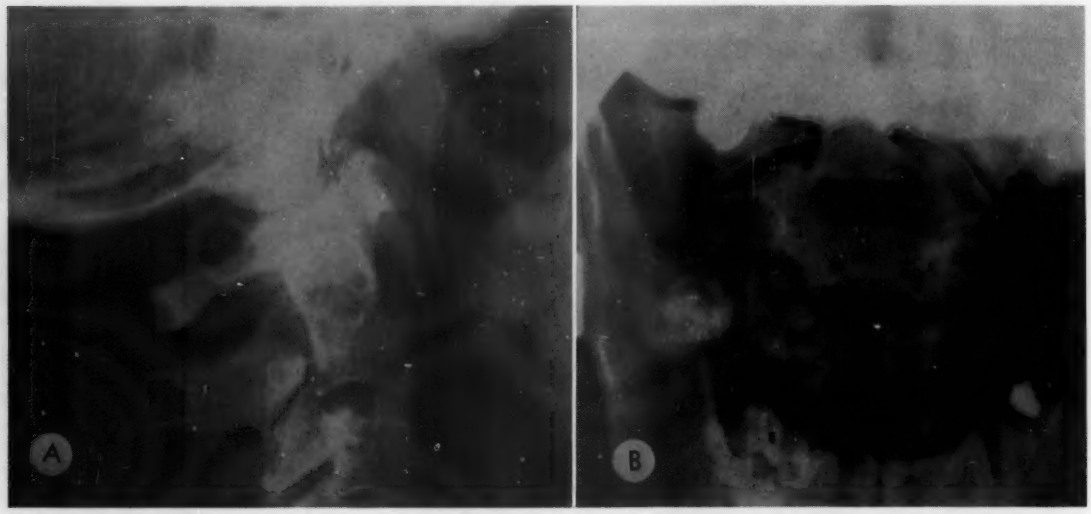

Fig. 17. (*A* and *B*) Asymmetry of the atlanto-axial facets. J.Q., male, aged fifty-nine, asymptomatic.

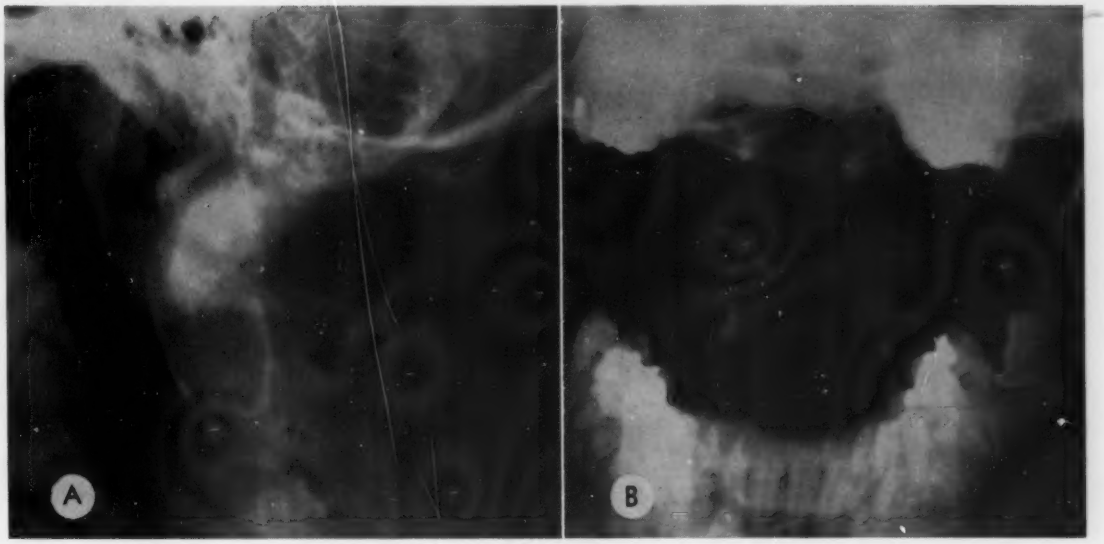

Fig. 18. (*A* and *B*) Fusion of C1 and C2. T.O., male, aged nineteen, asymptomatic.

with occipital neuralgia nor have I seen osteoarthritis of these joints in such a patient.

Complex fusions of the upper cervical vertebrae may, or may not, lead to central nervous system symptoms or signs. Each case must be judged individually (Fig 18, *A* and *B*, and 19). The demonstration of atlanto-axial dislocation or of a small effective foramen magnum in these cases indicates potential, if not actual, compression of the neuraxis.

In about half of our cases of occipitalization of the atlas, the second and third cervical vertebrae were fused. The presence of such a fusion did not correlate with the presence of neurologic symptoms or signs, however. When fusion of C2 and C3 is seen on skull or spine roentgenograms, one should examine the atlas carefully to ascertain that it is not fused to the skull.

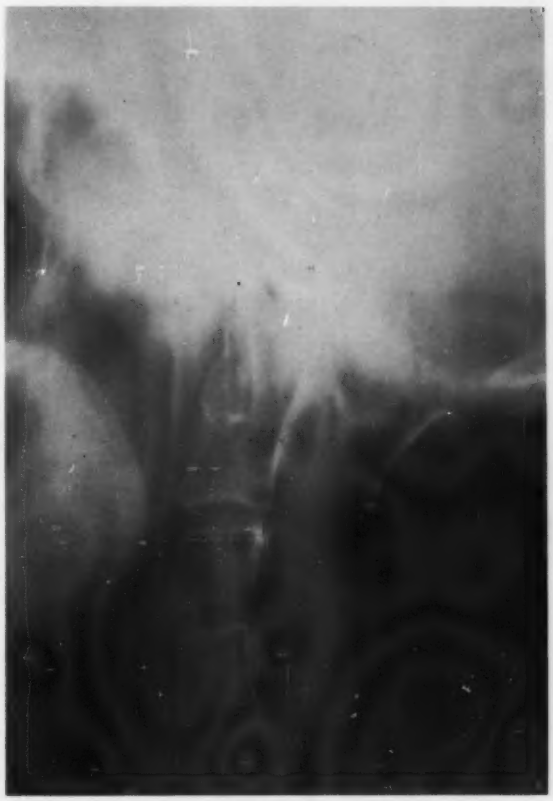

Fig. 19. Fusion of C1 and C2. S.G., male, aged thirty-seven. He had numbness of the right leg as well as pain and stiffness of the left arm for a year or more. Previously a diagnosis of multiple sclerosis had been made.

KLIPPEL-FEIL SYNDROME

The Klippel-Feil syndrome consists of the three signs, short neck, low hairline and restricted neck movement. It is graphically described by the phrase "L'homme sans cou."

It is said that this syndrome is due to

numerical reduction in cervical segments, but I am not convinced that this is always true. I have dissected the cervical spine in only one case of Klippel-Feil syndrome and in that case it was impossible to accurately determine the number of cervical vertebral bodies since there were no intervertebral disks. However, the cervical vertebral pedicles and cervical nerve roots were normal in number, although some of them were very small. In such a case, the etiology of the lesion is probably defective formation of alternating centra and intervertebral disks during the late blastemal and early cartilaginous stages of vertebral development. If the mesoblastic cells of the caudal halves of the sclerotomes do not proliferate normally, the intervertebral disk anlages may be small or absent and the chondrification centers of the centra may be continuous with each other. The anlages of the vertebral arches are dorsal outgrowths of the paraxial mesoderm of the sclerotomes between the myotomes. In the above mentioned specimen, these dorsal outgrowths must have formed in normal numbers. It is possible that the vertebral pedicles and the nerve roots are more accurate indicators of the segments of the spine than are the vertebral bodies. However, careful dissections of the cervical spine in more cases of Klippel-Feil syndrome are necessary to establish whether or not a normal number of cervical vertebral pedicles or nerve roots is present in all cases.

It is surprising that the Klippel-Feil syndrome is not always associated with central nervous system abnormalities since the spine and spinal cord developed together. The Klippel-Feil syndrome is often present in babies with occipital or cervical meningoceles, Arnold-Chiari malformations or myelodysplasias but such lesions are frequently fatal. Patients with the Klippel-Feil syndrome who survive to adulthood are usually free of central nervous system symptoms and signs. However, in these patients, hypermobility of the spine at levels adjacent to the fused segments may lead to excessive disk degeneration with posterior disk protrusion and secondary spinal cord disease. If the vertebral fusion extends very far into the thoracic region, it is usually accompanied by fusion of the ribs to the vertebrae. This may result in impaired pulmonary ventilation and abnormal susceptibility to respiratory infection.

NEURENTERIC CANAL LESIONS

The neurenteric canal is a transitory communication between the primitive gut, the chordal canal and the dorsal surface of the embryo. It is present for a few days in the third week of embryonic life. It is situated at a point which corresponds roughly to the cervical spine. Defective closure of the neurenteric canal or the presence of accessory neurenteric canals may result in an adhesion, a cyst or a canal connecting a part of the gastrointestinal or respiratory tract with the spine, the spinal cord or the dorsal surface of the fetus. In patients with posteriorly situated intrathoracic or upper abdominal tumors or cysts, the presence of anomalies of the cervical or upper thoracic vertebrae should lead to serious consideration of neurenteric cyst or re-duplication of part of the upper gastrointestinal tract.[5] This is especially important if the spinal anomaly involves the vertebral bodies or disks. The connection between anomalies of the cervical spine and anomalies in the upper abdomen may seem tenuous until one recalls that the anlage of the diaphragm is found in the cervical region in early embryonic life. The spinal anomaly may be severe and obvious but in some cases it is minor and easily missed, as in a case of a still-born child in whom a large posterior mediastinal cyst was found at autopsy (Fig. 20). The small stalk of this cyst passed through the rudimentary disk between the grouped hemivertebrae in the lower cervical region.

CERVICAL DISK DISEASE OR CERVICAL OSTEOARTHROSIS OR CERVICAL SPONDYLOSIS

Over the past ten years, there has been increasing interest in this condition be-

cause it has been shown that the pathologic changes in the spine may lead to disease of the cervical spinal cord as well as to pressure on cervical nerve roots. Some of these cases have been cured or improved by surgical treatment.

As far back as 1897, von Bechterew[7] described elderly patients with upper thoracic and cervical kyphosis, weakness and fasciculations of the muscles of the arms, legs, trunk and neck and loss of sensation in the lower cervical and thoracic dermatomes. They probably were instances of advanced spinal cord disease secondary to cervical spondylosis. It is likely that many such cases in the past have been diagnosed as von Bechterew's spondylitis, syphilitic pachymeningitis, progressive muscular atrophy, amyotrophic lateral sclerosis, subacute combined degeneration of the cord, etc. Even today, the differential diagnosis is sometimes difficult and we must accept the fact that some patients with early motor neurone disease will be treated surgically for cervical spondylosis. As our understanding of these conditions improves, such cases will become fewer and fewer.

Spondylosis is defined as ankylosis of a vertebral joint. The term is used mostly by neurologists and neurosurgeons for the condition formerly called osteoarthritis or osteoarthrosis of the spine in which ankylosis is usually not present. However, the terms "osteoarthritis" and "osteoarthrosis" are not entirely satisfactory since the osteophytes on the margins of the vertebral bodies are almost certainly secondary to traumatic and/or degenerative changes of the intervertebral disks and of the articular cartilages of the vertebral bodies. The term diskogenic disease of the spine is clumsy but probably the most accurate.

In this condition, osteophytes, *i.e.*, bony fringes, project outwards from the margins of the vertebral bodies, cracks and erosions are present in the articular cartilages of the vertebral bodies, desiccation and fibrillation of the nuclei pulposi have taken place; and there is bulging, or even rupture, of the annuli fibrosi (Fig. 21, *A* and *B*). It is important to emphasize that when osteophytes are seen projecting outward rather than upward and downward from the margins of the vertebral bodies there is always protrusion of intervertebral disk tissue of some degree in the same direction. When osteophytes and disks project posteriorly and posterolaterally from the vertebral bodies into the spinal canal or intervertebral foramina, they become potential causes of pressure on the spinal cord and cervical nerve roots. In Marie-Strümpell arthritis, the osteophytes on the margins of the vertebral bodies usually form in young patients in whom the disks are still normal.

Fig. 20. Anteroposterior roentgenogram of the excised spine and the posterior mediastinal cyst which had been filled with a radiopaque solution. The stalk of the cyst entered the rudimentary intervertebral disk in the center of the mass of fused hemivertebrae in the lower cervical region.

Fig. 21. Cervical spondylosis. (*A*) Lateral photograph and (*B*) roentgenogram of a thin slice through the sagittal plane of an excised cervical spine.

In such patients, the osteophytes project upward and downward, parallel to the normal course of annulus fibers, producing the bamboo spine with which we are all familiar.

Disk degeneration and protrusion may be present without osteophytes but osteophytes soon appear at the level of a ruptured annulus fibrosus or a herniated nucleus pulposus or at a level at which a discoidectomy has been carried out (Fig. 22).

Marked disk thinning and osteophyte formation are present at the C5–C6 and/or the C6–C7 levels in more than 50 per cent of spines in patients over forty years of age.[4] The frequency is about the same in asymptomatic patients as in those who have symptoms (Table VI). The most likely cause is the wear and tear of normal processes. This hypothesis is supported by the common observation of marked disk changes adjacent to fused segments of the cervical spine (Fig. 23, *A* and *B*). Presumably, the lack of motion at the level of the fusion leads to excessive movement at the adjacent level.

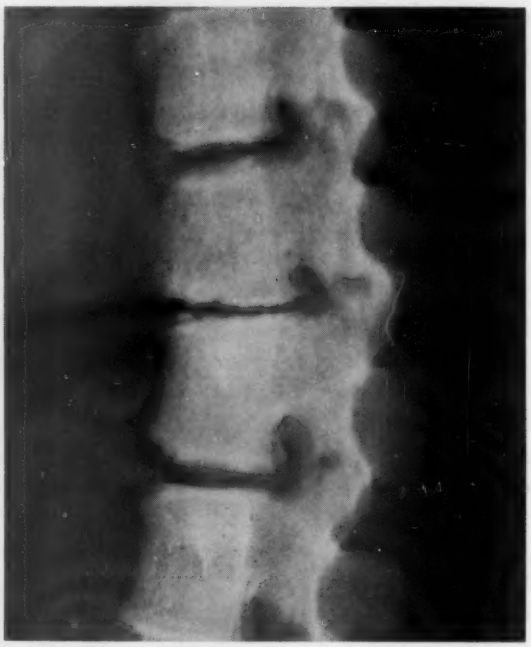

Fig. 22. Lateral roentgenogram of the spine of a monkey three and one-half months after removal of a nucleus pulposus by way of a small incision in the lateral aspect of the annulus fibrosus.

When the cervical vertebral bodies come together following degeneration or escape of the nucleus pulposus, their lateral edges almost touch (Fig. 24, *A* and *B*). This leads to much curving and some atrophy of the laterally placed annulus fibers. The lateral osteophytes follow these curves and, on coronal section of the spine, they resemble hooks (unci). The spaces between them are called unco-vertebral or Luschka joints (Fig. 25, *A* and *B*). I believe they are false

TABLE VI

FINDINGS ON CERVICAL ROENTGENOGRAMS OF 240 PATIENTS WITH AND WITHOUT SYMPTOMS RELATIVE TO CERVICAL SPINE OR CERVICAL SPINAL CORD[4]

Cervical Disk Disease	Age							
	30–39		40–49		50–59		60–Over	
	Symptomatic	Asymptomatic	Symptomatic	Asymptomatic	Symptomatic	Asymptomatic	Symptomatic	Asymptomatic
No. of Patients	10	48	37	54	28	22	15	26
Per Cent Normal Roentgenograms C2–T1	60	25	22	28	7	13	0	0
Per Cent Thin Disks Only	20	17	11	8	50	13	26	8
Per Cent Posterior Osteophytes Only	10	8	3	27	0	22	0	0
Per Cent Thin Disk and Posterior Osteophytes at Same Level	10	13	45	31	53	33	60	85

Note: In the different categories, the various pathologic findings appear as often in asymptomatic patients as in those who have symptoms.

FIG. 23. (*A*) Lateral roentgenogram and (*B*) lateral myelogram of the cervical spine of a patient with fusion of some of the upper cervical vertebrae and also fusion of some of the lower cervical vertebrae. The bending roentgenograms showed appreciable movement only at the level of the thin disk and the large osteophytes, and at this level the myelogram showed posterior projection of disk and/or bone.

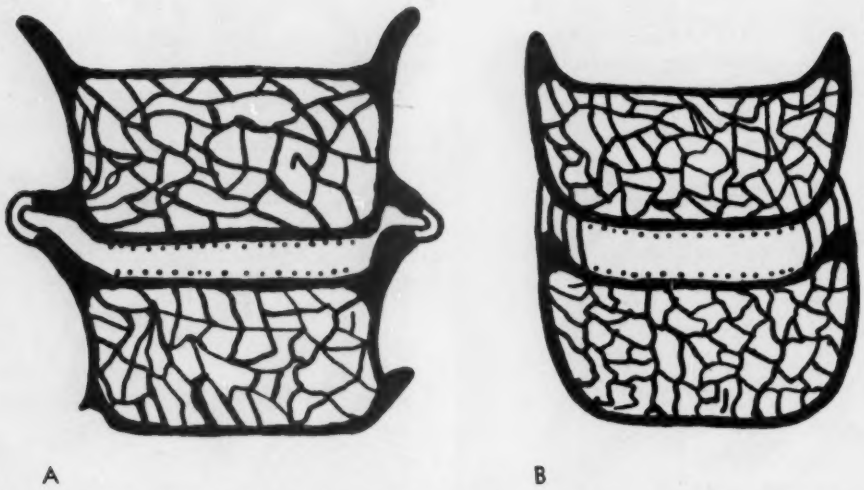

FIG. 24. (*A*) Diagram of coronal section of cervical vertebrae showing lateral bulging of the fibers of the annulus fibrosus and of the osteophytes on the corners of the vertebral bodies in a patient with degeneration of the intervening intervertebral disk. (*B*) The appearance of coronal section in a normal patient. The cartilage plates are represented by dotted lines.

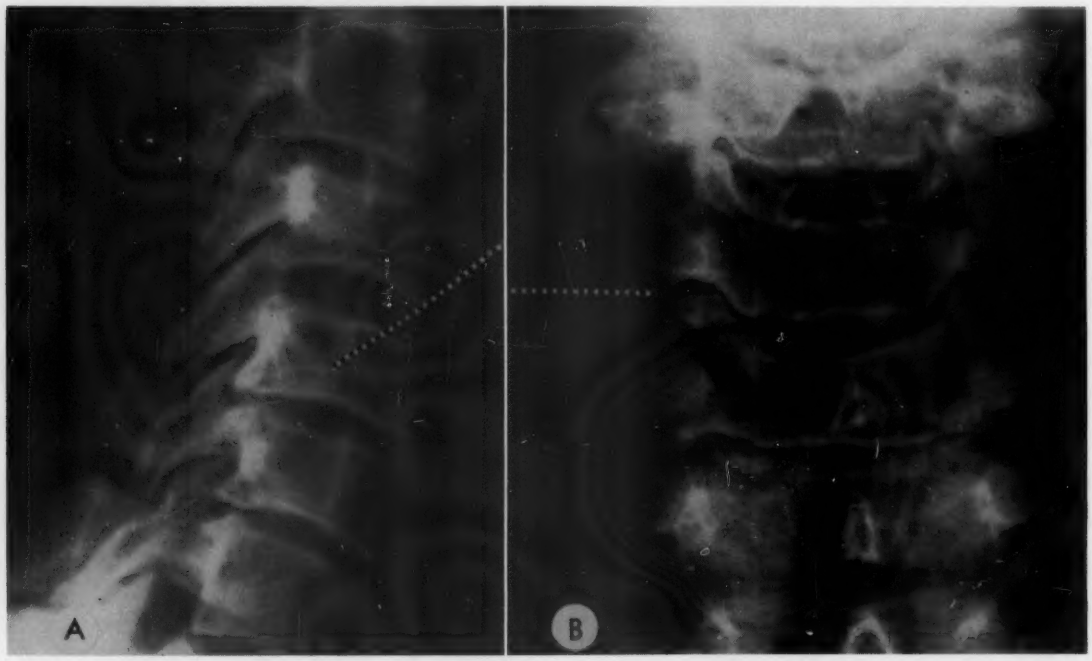

FIG. 25. (*A*) Lateral and (*B*) frontal roentgenograms of the cervical spine showing osteophytes (pointers) on the right margin of the bodies of C_4 and C_5 producing an unco-vertebral joint which extends from the anterior edge to the posterior edge of the lateral surfaces of these vertebral bodies. The curved gray line on the lateral roentgenogram (pointer) represents the space of the unco-vertebral joint. The lateral roentgenogram shows that these unci are projecting behind the plane of the vertebral body and therefore encroach on the intervertebral foramen.

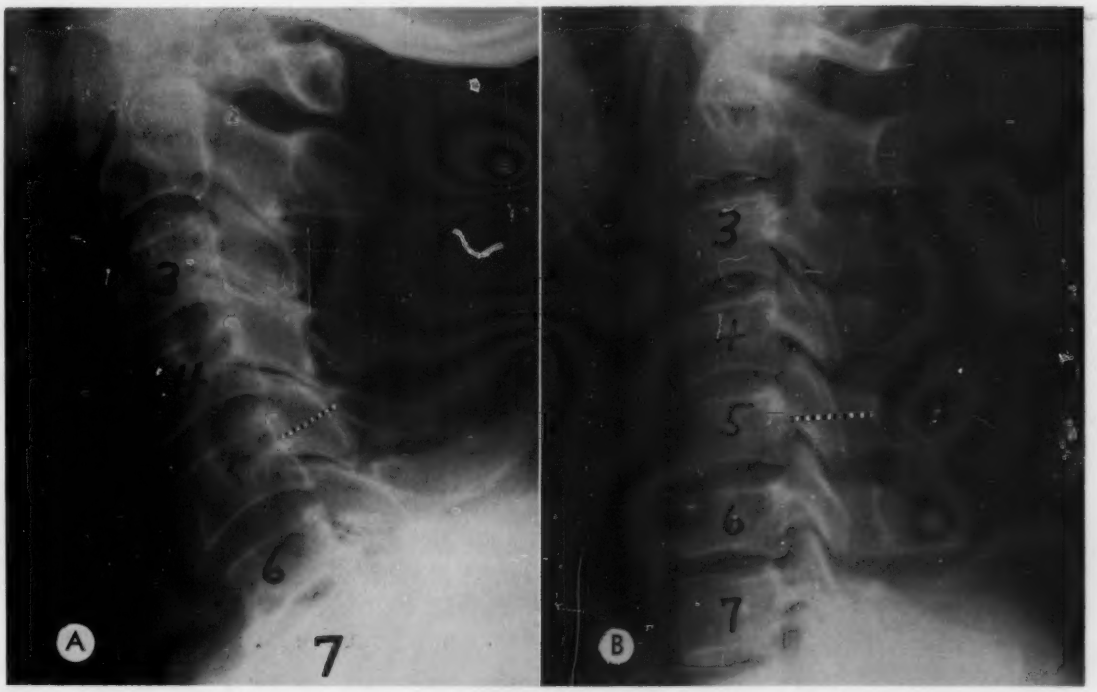

Fig. 26. Lateral cervical spine roentgenograms showing (*A*) a patient with a small anteroposterior diameter of the cervical spinal canal, and (*B*) a patient with a large anteroposterior diameter of the cervical spinal canal.

joints because they are most common at the lower cervical levels and are seldom, if ever, found in children or adolescents.

True osteoarthritis of the apophyseal joints of the cervical spine is almost as common as disk disease. Sometimes the apophyseal joint changes are present at levels where the disks are fairly normal, as is the case so often in the upper cervical region. In the lower cervical region, where disk disease is most frequently found, osteoarthritis of the apophyseal joints is rather rare. The lack of correspondence of degenerative changes in the anterior and posterior intervertebral joints is so common here that I doubt if disk disease is the primary cause of apophyseal joint disease. The practical importance of osteophytes on the margins of the articulating facets is that they may encroach on the spinal canal and on the intervertebral foramina. If apophyseal joint changes and disk disease are present at the same level, the spinal cord and the nerve roots here are in double jeopardy.

As the cervical vertebral bodies come together following disk degeneration or escape, the cervical laminae usually come together also. The upper edge of one cervical lamina may even come to lie inside the lower edge of the lamina above it. This results in shortening and folding of the ligamenta flava and, perhaps, degeneration and swelling of these ligaments. Infolded or swollen ligamenta flava encroach on the posterior part of the spinal canal and must be considered as another potential cause of pressure on the cervical spinal cord. This is fairly common in the mid-cervical region, less so in the lower cervical region and rather rare at upper cervical levels.

The size of the bony spinal canal must also be considered. In patients with achondroplasia, there is generalized smallness of the spinal canal but usually this is most marked in the thoracic region, producing spastic weakness of the legs but not of the arms. In Paget's disease, fibrous dysplasia and certain other bone diseases, involve-

FIG. 27. Lateral cervical spine roentgenogram showing evidence of spondylosis (not marked) at the C_4–C_5 level as well as high-grade occipitalization of the atlas. Case L.F., male, aged sixty-two.

ment of a vertebral arch as well as a vertebral body can lead to localized smallness of the spinal canal and local constriction of the spinal cord. More common is developmental smallness in the anteroposterior diameter of the cervical spinal canal, a variation of normal. For practical purposes, this can be determined when the posterior wall of the spinal canal is close to the plane of the posterior surfaces of the articular pillars of the cervical vertebrae (Fig. 26, *A* and *B*). All of the cervical vertebrae may be involved or only a few. The fourth, fifth and sixth cervical vertebrae appear to be affected most often and to the greatest

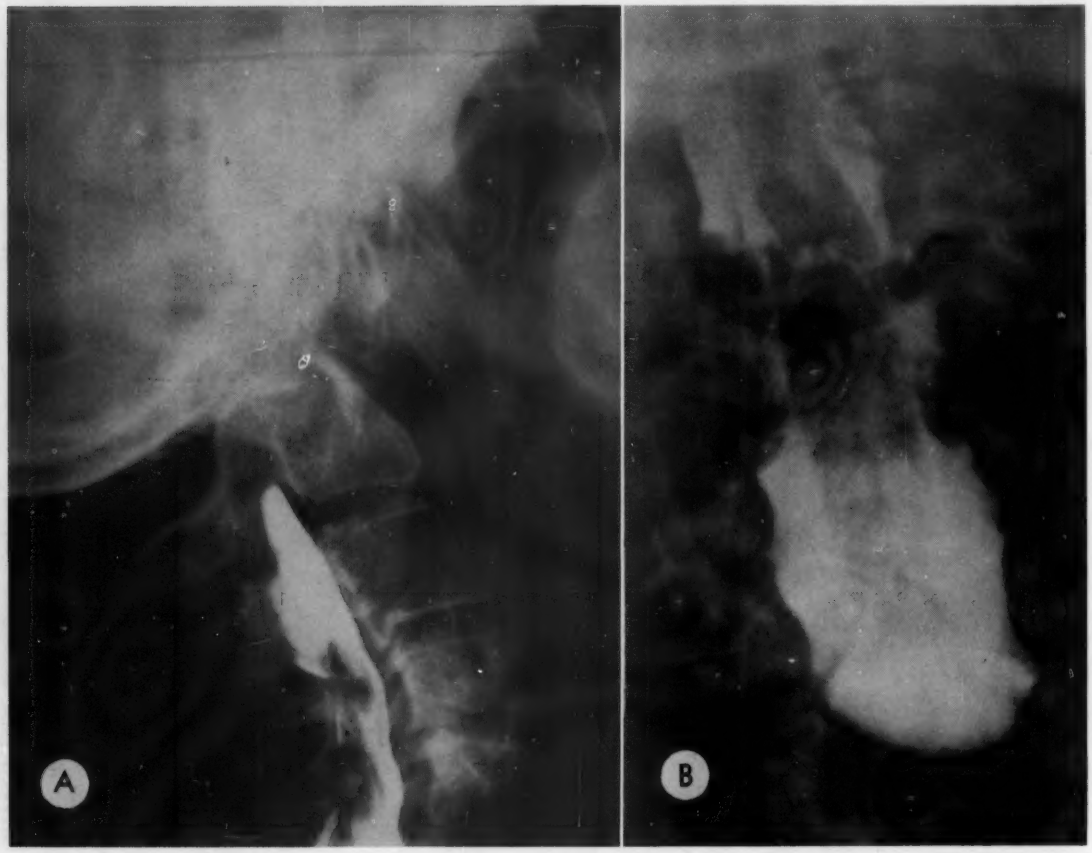

FIG. 28. (*A* and *B*) Myelograms showing posterior projection of disk and bone at the C_4–C_5 and C_5–C_6 levels and also anterior bulging of the ligamenta flava at the same levels. Same patient as in Figure 27.

Fig. 29. Photograph of the cerebellum, medulla oblongata and spinal cord of the patient shown in Figures 27 and 28. Note the constriction of the lower medulla oblongata and the absence of ridging of the anterior surface of the spinal cord in the mid-cervical region.

degree. Payne and Spillane[6] have shown that patients suffering from cervical spondylosis with paraplegia have a significantly smaller spinal canal in the anteroposterior diameter than normal patients or even those with spondylosis who have no paraplegia. This agrees with our findings at the Montreal Neurological Institute and we believe that a small spinal canal at one or more levels in the cervical region is an important contributory cause of cervical cord disease of this nature.

Besides the factors mentioned above, there are variations in the arteries and veins of the spinal cord and in the tightness or looseness of the dentate ligaments that

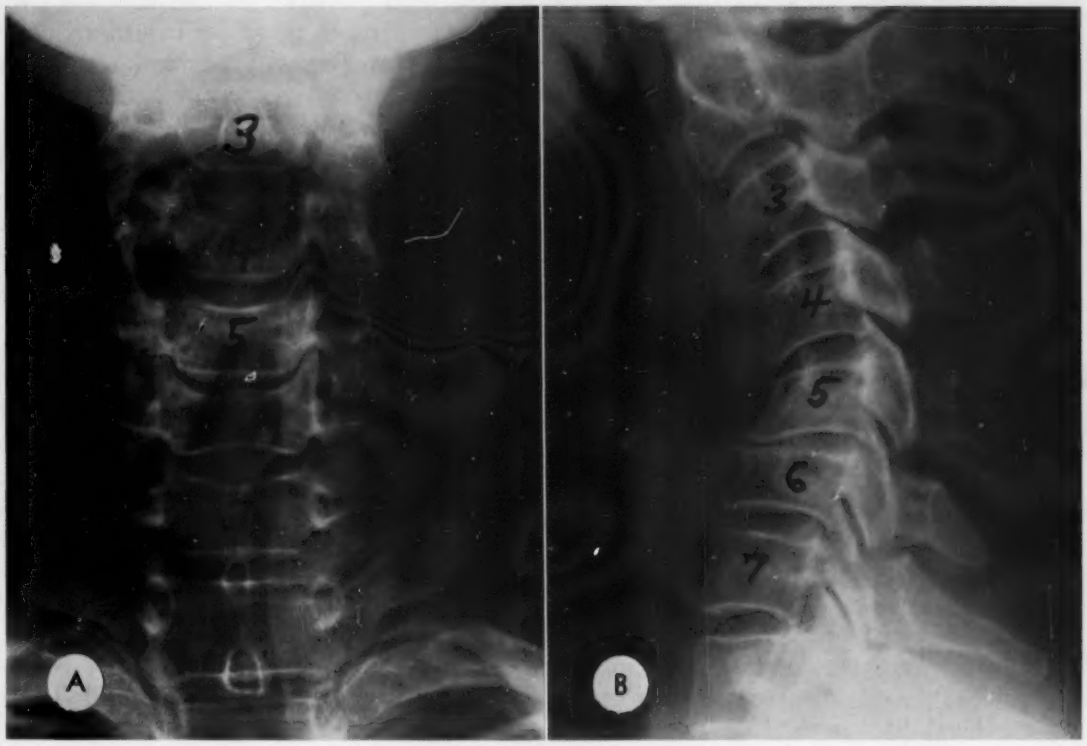

Fig. 30. (*A* and *B*) Cervical spine roentgenograms showing the C5–C6 disk to be thin and surrounded by osteophytes.

FIG. 31. Myelogram of the patient in Figure 30 showing a large unilateral posterior protrusion of the C6–C7 disk with no myelographic evidence of posterior protrusion of disk or bone at the C5–C6 level.

must also be considered. The latter cannot be assessed roentgenologically.

The relationship of the neurologic symptoms and signs to the abnormalities of the bones, joints and ligaments in cervical spondylosis has not been worked out completely. In the present state of uncertainty, I think we must place our major dependence on three things.

First, and most important, the level of neurologic signs must correspond closely to the level of pathologic changes shown roentgenologically. Since more than 50 per cent of all patients over the age of forty have cervical spondylosis, its presence must not blind us to other lesions. Figure 27 shows the spine of a sixty-two year old man with a history of progressive weakness of the legs for five years and of the arms for several months. Cervical spondylosis at the C4–C5 level had been diagnosed and he had been sent to the Montreal Neurological Institute for surgical treatment. His myelogram (Fig. 28, *A* and *B*) showed some posterior projection of disk and bone, as well as anterior bulging of the ligamenta flava at the C4–C5 and the C5–C6 levels. However, roentgenograms also showed occipitalization of the atlas with the odontoid process lying inside the foramen magnum. This was thought much more likely to be the cause of severe neurologic signs. At operation, the medulla oblongata was found to be greatly compressed, while the spinal cord at the fourth, fifth, and sixth cervical vertebrae was compressed little, if any. Unfortunately, he died twelve days after operation from pulmonary infarction. Autopsy showed narrowing of the neuraxis at the foramen magnum but no gross changes at the C4–C5 or the C5–C6 levels (Fig. 29). Histologic examination revealed that the changes in the spinal cord began at the foramen magnum. They were about the same in the upper cervical region as in the lower cervical and thoracic regions.

Secondly, the effects of pathologic changes lateral and posterior to the cervical cord and nerve roots must be considered. Although posterior and posterolateral disk protrusions and posterior and posterolateral osteophytes are the most common causes of pressure on the cervical cord and nerve roots, they are not the only factors. Striking transverse grooves on the anterior surface of the spinal cord are occasionally seen at autopsy in patients who have had few, if any, neurologic signs or symptoms. Their spinal cords probably escaped damage because there was very good blood supply or because there was no counter pressure from behind or because the dentate ligaments were loose, letting them move backward. Their cervical nerve roots probably escaped damage, although there were osteophytes in the intervertebral foramina, because there was only slight protrusion of disk tissue into the same foramina.

Thirdly, ordinary cervical spine roentgenograms alone are not enough. These show chronic changes such as thin disk and osteophytes but often show no evidence of recent lesions (Fig. 30, *A* and *B*, and 31).

Cervical myelography using sufficient oil to fill the cervical spinal canal completely or almost completely must be carried out before final and complete roentgenologic opinion is given.

CONCLUSIONS

Congenital anomalies of the cervical spine, cervical disk disease, osteoarthritis of cervical apophyseal joints and smallness of the cervical spinal canal are frequently observed. Mere diagnosis of these conditions is not very helpful because they are present about as often in asymptomatic as in symptomatic patients. There are some pathologic factors and combinations of factors that are of considerable help in deciding when a condition is producing symptoms and signs. However, careful correlation of the neurologic with the roentgenologic findings remains the best approach when assessing any individual patient.

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REFERENCES

1. List, C. F. Neurologic syndromes accompanying developmental anomalies of occipital bone, atlas and axis. *Arch. Neurol. & Psychiat.*, 1941, *45*, 577–616.
2. McRae, D. L., and Barnum, A. S. Occipitalization of atlas. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *70*, 23–46.
3. McRae, D. L. Bony abnormalities in region of foramen magnum: correlation of anatomic and neurologic findings. *Acta radiol.*, 1953, *40*, 335–355.
4. McRae, D. L. Asymptomatic intervertebral disc protrusions. *Acta radiol.*, 1956, *46*, 9–27.
5. Neuhauser, E. B. D., Harris, G. B. C. and Berrett, A. Roentgenographic features of neurenteric cysts. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *79*, 235–240.
6. Payne, E. E., and Spillane, J. D. Cervical spine; anatomico-pathological study of 70 specimens (using a special technique) with particular reference to problem of cervical spondylosis. *Brain*, 1957, *80*, 571–596.
7. von Bechterew, W. Von der Verwachsung oder Steifigkeit der Wirbelsäule. *Deutsche Ztschr. Nervenh.*, 1897, *11*, 327–337.
8. Watson-Jones, R. Fractures and Joint Injuries. Fourth edition. E. & S. Livingstone, Edinburgh and London, 1952.

THE WIDTH (THIRD DIMENSION) OF THE SELLA TURCICA

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THE lateral roentgenographic view of the skull yields the length and the height (depth) of the sella turcica; the width (third dimension) of the sella has not yet been roentgenographically reproduced. Farinas[3] suggested measuring the width of the dorsum sellae as seen in the semiaxial (Towne's) view; also Bloch and Joplin[1] used the dorsum to evaluate the transverse diameter of the sella and the pituitary gland. The dorsum, however, is not really suitable for this purpose. In the semiaxial view of the skull as Mahmoud[5] stated: " . . . the rays used are oblique and the resulting picture distorted." Furthermore, the dorsum varies greatly in its coronal diameter; generally, it is broad at its upper end and slightly constricted inferiorly (Fig. 1*A*); the reverse, however, may also be true (Fig. 1*B*). Finally, when the sella enlarges, erosions of its dorsum may take place but the width of the dorsum does not increase. The dorsum will not, therefore, give us information regarding the width of the sella in pathologic cases.

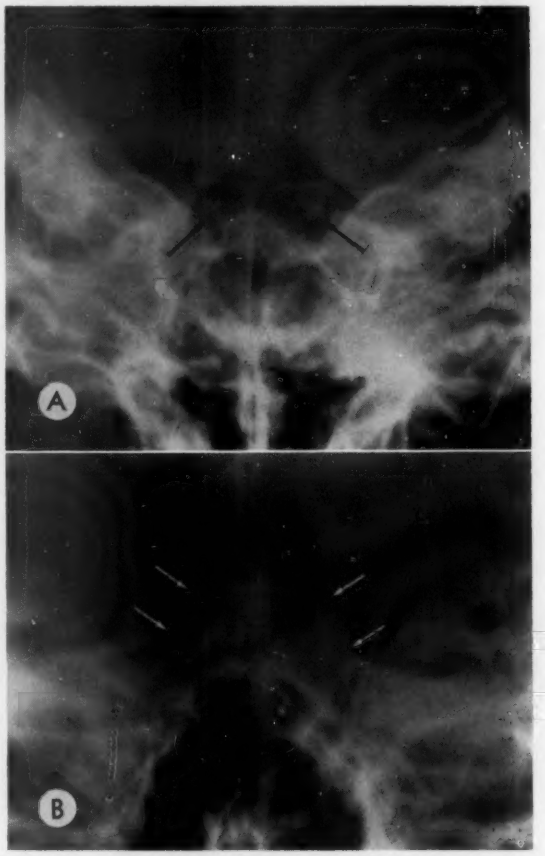

FIG. 1. (*A*) The dorsum is broad at its upper end and constricted inferiorly. (*B*) The dorsum is broader inferiorly.

MEASUREMENT OF THE WIDTH OF THE SELLA

The width of the sella may be determined on the standard posteroanterior roentgenograms of the skull. The floor of the sella is clearly seen and it may be delimited laterally thus yielding its width (Fig. 2, *A* and *B*). The floor of the sella is recognized in the posteroanterior views of the skull in over 90 per cent of the cases; with laminagraphy the floor is visualized in 100 per cent of the cases. In most instances the coronal outline of the sellar floor appears as a "plateau." This plateau is flat, or concave, rarely convex. Figure 3 shows tracings of the "coronal" sellar outline in 100 "normal" adults; in some the contours of the sphenoid sinus have been included. The 100 tracings have been obtained from roentgenograms of 108 cases picked at random in our film library. Fairly frequently two short sloping edges connect the main plateau of the floor with the lateral walls of the sphenoid sinus (Fig. 4*A*); on these slopes—carotid sulcuses—lie the carotid syphons (Fig. 4*B*). In almost every

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case the lateral edges of the plateau are demonstrated. The floor of the sella is clearly recognized also in the posteroanterior views of the skulls of young children (Fig. 5).

Measurements of the width of the sellar floor may be easily obtained in the great majority of cases. The landmarks used for the measurements are the highest points of the lateral edges of the aforementioned plateau. In a few cases, in particular when the coronal outline of the floor of the sella is convex, the choice of landmarks for measuring the width of the sella may be difficult; in these cases the present author suggests measuring the distance between two ideal lines continuing cranially from the lateral walls of the sphenoid sinus. Figure 6 is a barograph of the width of the sella as determined in 100 "normal" adult cases. Fifty per cent of the cases show a width of 13 or 14 mm.; 2 cases show a width of 9 mm. and only 1 case a width of 18 mm. Measurements in 10 children under five years of age showed a width of 10 mm. in 2 cases, 11 mm. in 3, 12 mm. in 1 and 13 mm. in 4. The above figures have not been corrected for the roentgenographic magnification; this has been calculated to be about 10 per cent (36 inches focal distance).

When the sella enlarges, the width of the sella enlarges too (Fig. 7, *A* and *B;* 8, *A* and *B;* and 9, *A* and *B*). The width of the sella may reach 20, 25, up to 30 mm. Generally, there is correspondence between the increase in length and height and the increase in width; the sella, however, may enlarge in only one of its diameters while the others remain within normal limits. In addition to increase in its diameter, the "frontal plateau" of the sella almost invariably becomes concave, vaguely resembling a saucer (Fig. 10, *A* and *B;* and 11, *A* and *B*).

Fig. 2. The floor of the sella as seen on a standard posteroanterior roentgenogram. The floor may be delimited laterally thus yielding the width of the sella. In *A* the central beam is less inclined than in *B*. The floor is clearly seen in both instances.

ADVANTAGES OF THREE-DIMENSIONAL STUDY OF THE SELLA

The following advantages are offered by a three-dimensional study of the sella: (1) better evaluation of the enlarged sella; (2) recognition of the floor of the sella in the frontal plane when it is not clearly seen on the lateral roentgenogram of the skull (floor hidden by extensive mastoid pneumatization or not seen because markedly deepened); (3) objective study of the asymmetric sella; (4) better evaluation of the early erosion of the cortical lining of the sellar cavity due to increased intracranial pressure; (5) accurate knowledge of the lateral boundaries of the sella (particularly important if transphenoid surgery is con-

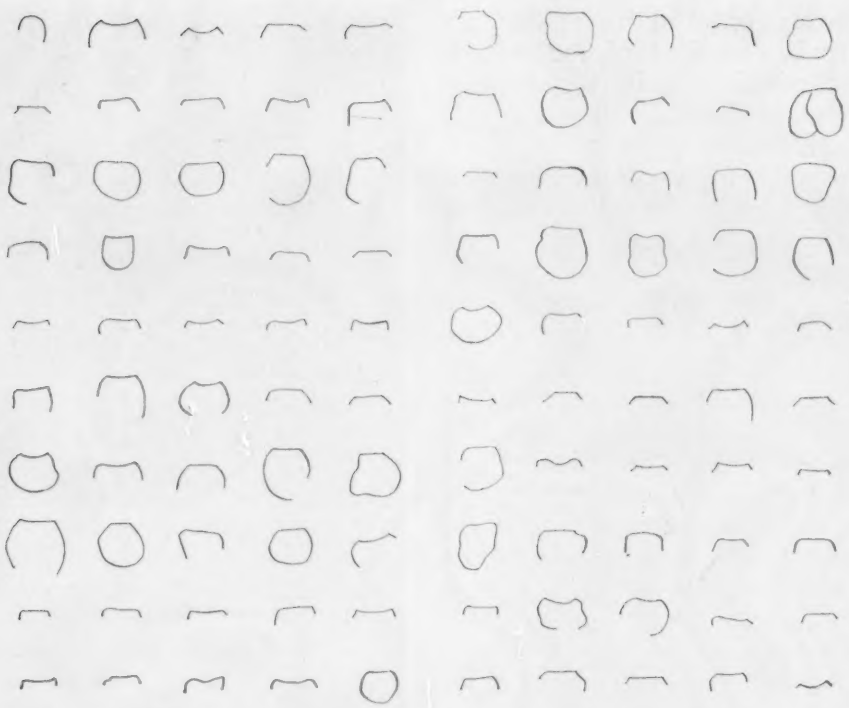


FIG. 3. The "coronal" sellar outline in 100 cases of "normal" adults.

templated); (6) the borderline sella, an everyday neuroroentgenologic problem, is more adequately evaluated.

Sellas considered enlarged on the lateral roentgenogram may prove to be only long and narrow, but within normal limits (Fig. 12, *A* and *B;* and *C* and *D*). On the other hand sellas considered normal may be abnormally wide and therefore pathologic (Fig. 13, *A* and *B*). The so-called "small sellas" may prove to be normal, or in exceptional cases enlarged.

VOLUMETRIC EVALUATION OF THE SELLA AND OF THE PITUITARY GLAND

Mahmoud[5] proved in his valuable monograph that the length and height of the sella correspond to the length and height of the pituitary gland. The width of the sella corresponds to the width of the pituitary gland. Our anatomic studies support this conclusion (Fig. 14, *A* and *B;* and 15, *A* and *B*).

Until now only linear or area measurements of the sella have been possible. In his textbook on neuroradiology Orley[7] states: "The radiographic measurement of the sella in the lateral view of the skull is not . . . an absolute guide to the dimensions of the hypophysis, as the lateral view of the sella only indicates the length and depth of the hypophysis but gives no idea of its width." The fact that now the three diameters of the sella may be obtained makes possible an evaluation of the volume of the sella. Information regarding the size of the pituitary gland may also be gained. It should be clear, however, that there are some limitations in obtaining the volume of the pituitary gland from the volume of the sella. Already in 1924 Rasmussen[8] noticed that there may be a wide variation between the bony compartment and the weight of the enclosed hypophysis. The dura surrounding the pituitary which is, of course, contained within the sella may in a few instances represent as much as 25 per cent of the total weight of the intrasellar contents. Pockets of subarachnoid space

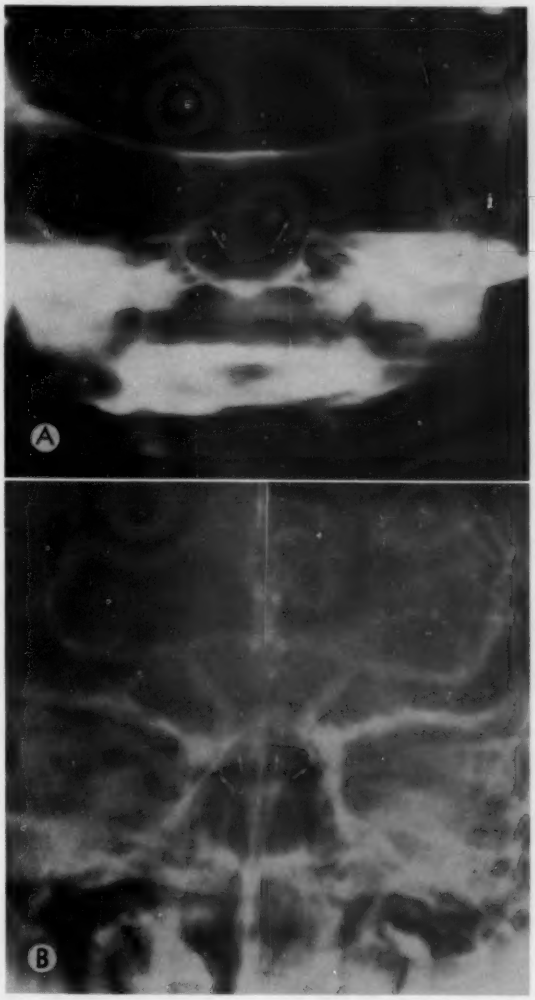


Fig. 4. (*A*) The carotid sulcuses. (*B*) The carotid syphons lying on the carotid sulcuses.

may be contained within the sellar cavity. These pockets which are occasionally quite sizable were first described by Hughson[4] in 1922, and have been confirmed by Engels[2] in 1958 and by Oppenheimer[6] in 1959. Incidentally Ergels and Oppenheimer could demonstrate encephalographically the intrasellar extension of the subarachnoid space. We have also found that subarachnoid pockets may fill a great part of the sellar cavity (Fig. 16, *A* and *B*). Considering therefore that the sella does not contain only the pituitary gland proper with its three portions and its capsule but also the covering meninges and sometimes

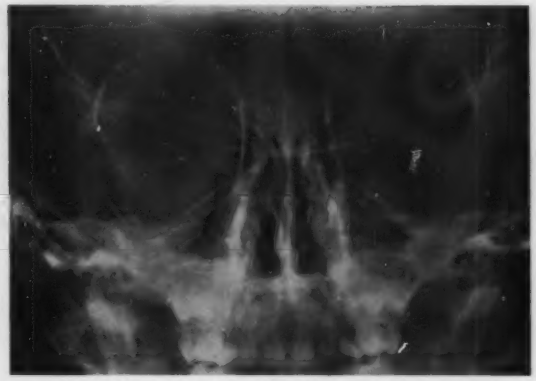


Fig. 5. The floor is clearly seen on a posteroanterior roentgenogram of the skull of a three year old child.

extensions of the subarachnoid space, it is clear that assessing the volume of the pituitary gland from the volume of the sella must be done with caution. On the other hand part of the pituitary gland, a section of the stalk, is, of course, above the diaphragm and outside the sella; the stalk may account for 5 per cent of the weight of the pituitary gland. Figure 17 shows the different components with the relative percentile weights of the pituitary gland. In addition to these normal anatomic characteristics, certain pathologic anatomic features likewise limit the value of appraising the size of the pituitary gland from the volume of the sella. The sella may enlarge independently from an increase in the volume of the pituitary gland. A dilated third ventricle, parasellar tumor and aneu-

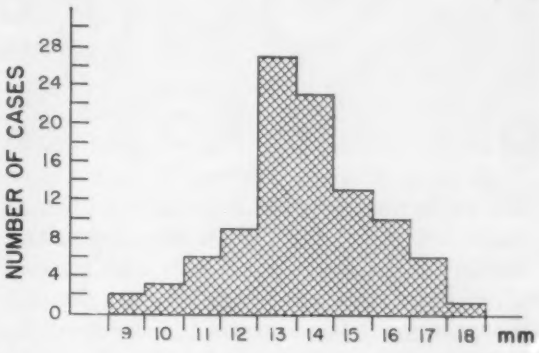


Fig. 6. Width of the sella in millimeters as determined in 100 "normal" adult cases: no magnification correction.

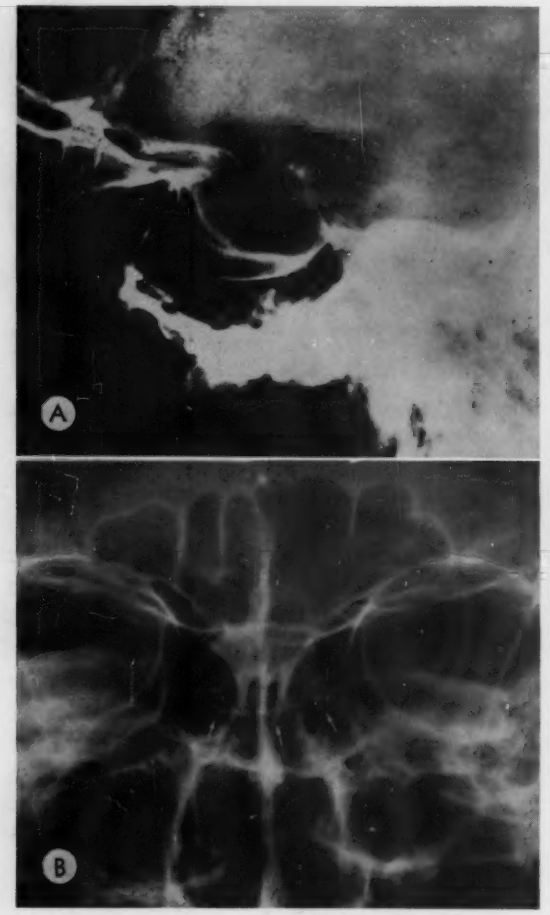


Fig. 7. (*A*) Verified chromophobe adenoma. (*B*) The floor of the sella in the frontal view.

rysms, general increased intracranial pressure, tumor of the epipharynx and sphenoid sinus may all cause sellar enlargement. Not all enlarged sellas are therefore due to enlarged pituitary glands, the corollary to this statement being that an enlarged sella does not necessarily mean an enlarged pituitary gland. On the other hand, the pituitary enlargement must be significant to cause a sellar enlargement. Apart from the adenomas arising from the pituitary tissue located above the diaphragma sellae, intrasellar pituitary adenomas with normal sellas have been reported. The weight and the volume of the pituitary gland markedly increase during pregnancy, reach their maximum at the end of pregnancy, and then sharply decline. The sella, however,

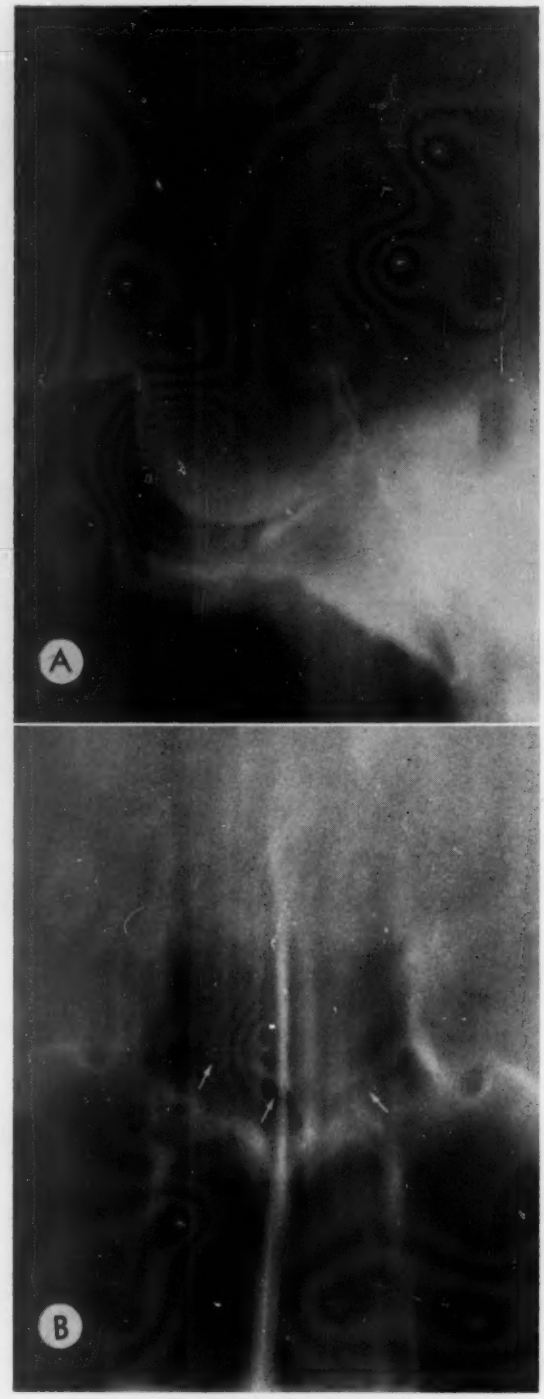


Fig. 8. (*A* and *B*) Laminagrams showing a nonverified intrasellar tumor.

does not increase in size during pregnancy; we have checked this point in 2 cases. In summary, there are limitations to be con-

Fig. 9. (*A* and *B*) Laminagrams showing a verified intra- and suprasellar craniopharyngioma.

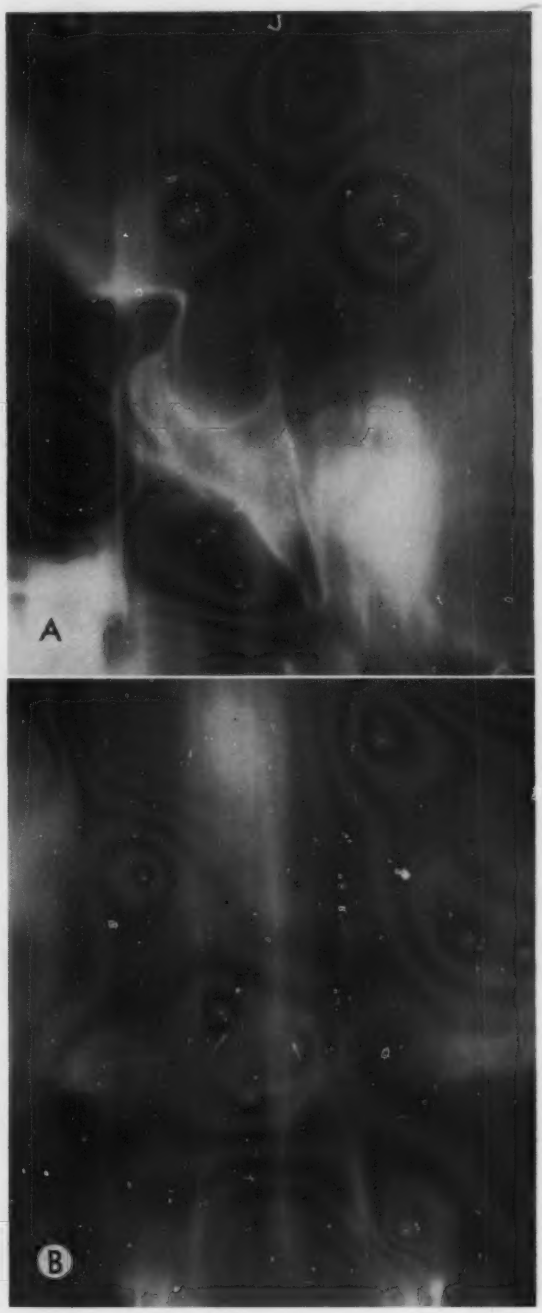


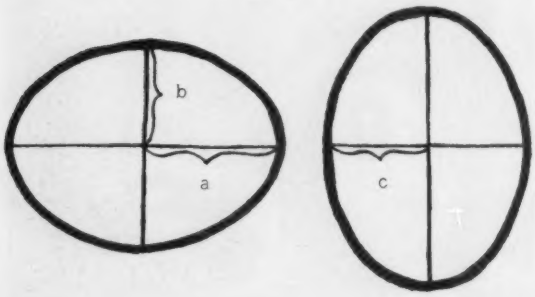
Fig. 10. (*A* and *B*) Laminagrams showing a verified basophilic adenoma. In the frontal view the sella is "saucer-shaped."

sidered when appraising the pituitary gland volume from the volume of the sella. These limitations derive from the following facts: (a) the intrasellar content is not due to the pituitary gland proper only but also to its coverings, (b) part of the pituitary gland is extrasellar, (c) the sella may enlarge without enlargement of the pituitary gland for various pathologic reasons, and (4) the pituitary gland may enlarge without caus-

ing enlargement of the sella. How big the pituitary enlargement must be to cause enlargement of the sella remains to be seen. Despite all of the above limitations, we believe that a roentgenographic volumetric appraisal of the pituitary gland may be very worthwhile and undoubtedly more valuable than linear and area measurements suggested and used until now.

To calculate the volume of the pituitary gland, we assume that its shape is an ellipsoid and therefore we use the following formula:

ELLIPSOID



($\pi = 3.1416$)

Volume, $V = (4\pi/3)\ abc$

$= 4.1888\ abc$

or, if $b = c$,

$V = 4.1888\ ab^2$

where a, b, and c are ½ the length, the width, and the height.

MATERIAL AND METHOD

We thus far have studied 15 specimens of "normal" sellas and pituitary glands. Table 1 shows the obtained measurements in 10 out of these 15 cases. For these measurements the pituitary gland with its capsule and part of the stalk, without the dura, has been used. The volume determination has been made with the method of fluid displacement. The shrinkage of the pituitary due to formalin has been considered, checked and found to be negligible; this is in agreement with what Rasmussen[8] found in 1924. The average error in the volume determination from roentgenograms *in vivo* in 10 cases is 18 per cent determined by the values of actual measurement. We have been somewhat surprised that our value

TABLE I

Patient	Roentgenographic Volume in Vivo (magnification correction)	Rentogenographic Volume of Sella Turcica Specimen (no magnification correction)	Actual Pituitary Gland Volume (no dura, with capsule and part of stalk)
R.T. ♀43 Heart condition		0.653	0.658
C.H. ♂30 Leukemia	0.337		0.3893
F.R. ♂60 Heart condition		0.564	0.6284
L.H. ♀73 Leukemia	0.577		0.7241
J.W. ♂18 Leukemia	0.452		0.4728
B.B. ♂43 Heart condition		0.352	0.4721
S.E. ♂45 Lymphoma	0.625		0.7414
S.M. ♀67 Carcinoma of breast		0.586	0.6840
J.T. ♀33 Brain tumor	0.440		0.5910
C.F. ♀52 Carcinoma of cervix	1.031		0.8647

determination from roentgenograms was erroneous in the negative direction in 9 out of 10 cases. Considering that we have not included the dural covering of the pituitary

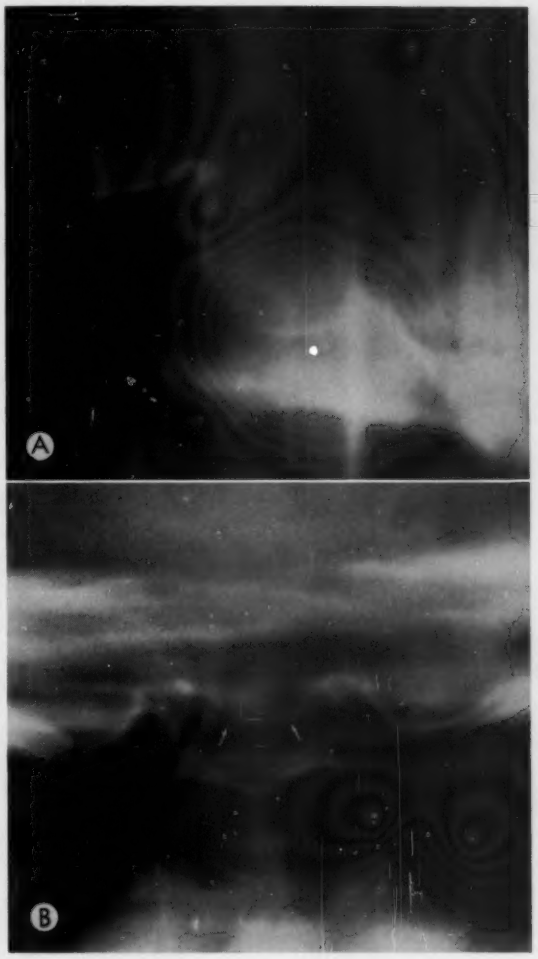


Fig. 11. (*A* and *B*) Laminagrams showing a nonverified intrasellar tumor. Again the sella is "saucer-shaped" in frontal view.

Fig. 12. (*A* and *B*) Roentgenograms of sella turcica specimen. The sella appears large in the lateral view; in the frontal plane it is, however, very narrow.

gland in our volume determination of the pituitary gland specimen, one might have expected an error in the positive direction. The explanation for this may be due to the fact that the pituitary gland is not perfectly ellipsoid and so the peripheral bulgings and irregularities may account for the error in the negative direction.

Regarding the choice of the different points which we have used as landmarks for our measurements, we have come to the following conclusions:

1. In measuring the length of the sella, the most distant points of the cortical outline of the anterior wall of the sellar cavity and the anterior aspect of the dorsum sellae should be used. When the anterior aspect of the dorsum shows a double cortical contour, the posterior cortical contour should be chosen.

2. In determining the height of the sella, the distance from the middle point of a line drawn between the tip of the tuberculum sellae and the tip of the dorsum sellae to the lowest point of the sellar floor should be used (Fig. 18).

3. For measuring the width of the floor, the distance between the two highest points located on the edges of the aforementioned plateau should be measured.

A simple method to measure the volume of the pituitary gland from the volume of the sella that can be used in everyday practice is based on the calculations summarized in Table II. Measurements of the length, width, and height of the sella obtained from roentgenograms are without

FIG. 12. (*C*) The sella after removal of the pituitary gland. (*D*) The removed pituitary gland with a very unusual "trapezoid" shape.

FIG. 13. (*A* and *B*) Nonverified intrasellar tumor. The sellar width has increased more than the other two diameters.

any correction multiplied; the obtained figure is divided by 2,000 giving us the estimated volume in milliliters. Figure 13, *A* and *B* shows the sella of a markedly acromegalic patient; the measurements of this sella are: length 11 mm., width 20 mm., and height 11 mm. By applying the above described simple calculation method, the

TABLE II

CORRECTION FACTORS BETWEEN ROENTGENOGRAPHIC VOLUME AND ACTUAL PITUITARY GLAND VOLUME

Correction factor K. = .5112 includes:

(1) correction factor from roentgenographic volume to actual pituitary gland volume

(2) magnification correction

and (3) correction for $4\pi/3\times(\frac{1}{2})^3$.

K. $\approx .5 = \frac{1}{2}$ is applied to the original roentgenographic dimensions. Therefore, the estimated volume is given by:

actual pituitary gland volume $= \frac{1}{2}(L\times W\times H)/1000$ ml.

where

L = length in mm.
W = width in mm.
and H = height in mm.

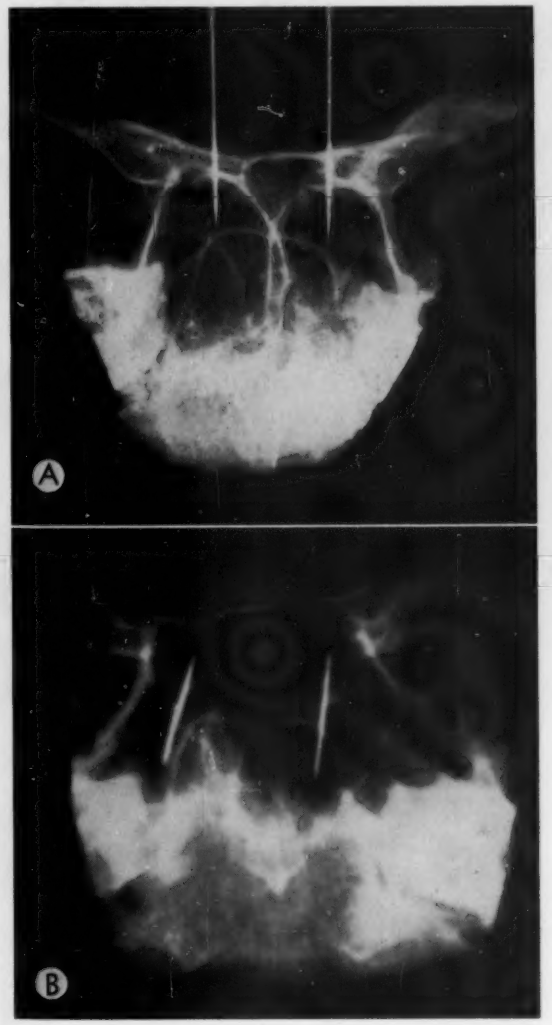


FIG. 14. (*A*) Two pins outline laterally the pituitary gland. Its width corresponds to the width of the sellar floor. (*B*) Pins lie horizontally close to the lateral aspects of the pituitary gland. Again the width of the pituitary gland corresponds to the width of the sella.

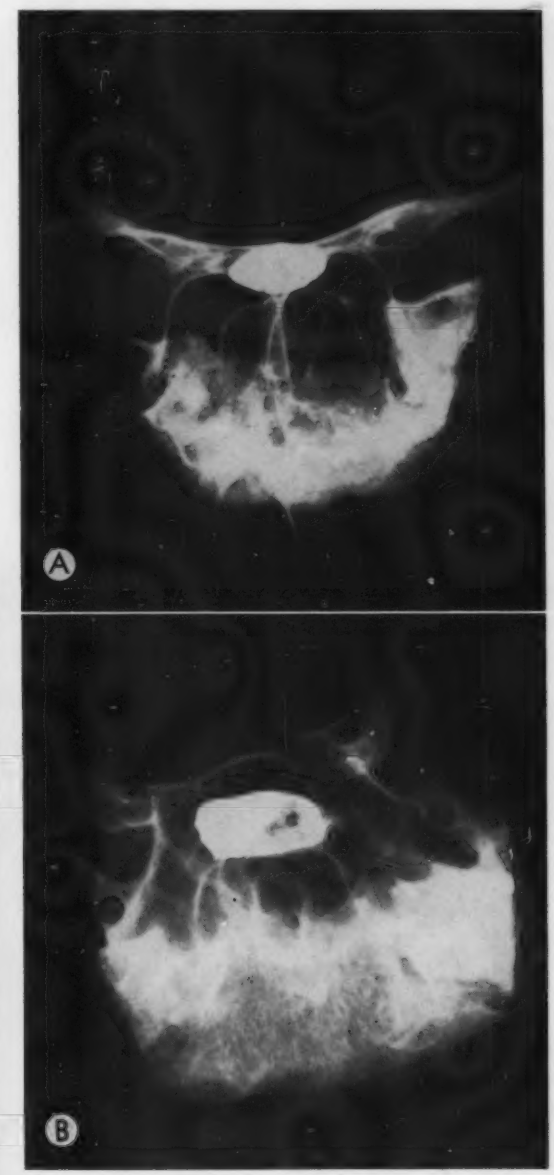


FIG. 15. (*A* and *B*) Two pituitary glands are thinly coated with an opaque solution. Their width corresponds to the width of the sellas.

Fig. 16. (*A*) A great part of the sellar cavity is filled with a huge subarachnoid pocket. (*B*) The same sella as in *A* with the dorsum "turned over."

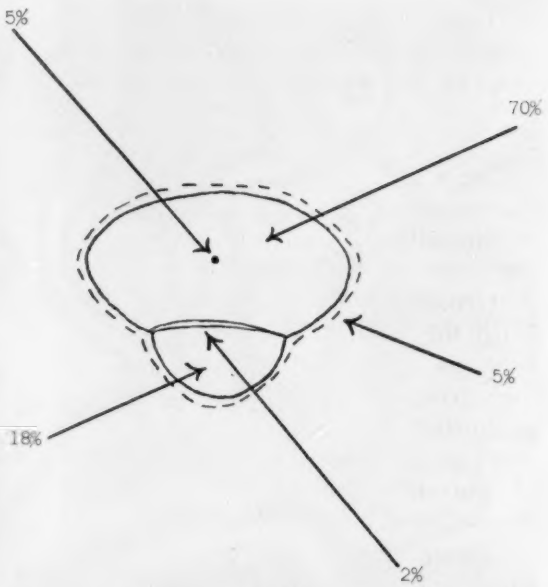


Fig. 17. The percentile weight of the different components of the pituitary gland: Seventy per cent anterior part; 18 per cent posterior part; 2 per cent intermediate part; 5 per cent stalk; 5 per cent capsule.

volume of the pituitary of this patient will be 1.21 ml., *i.e.*, definitely pathologic (the average volume of the pituitary in adults is 0.6 ml.). It is interesting to note that in this particular case the sellar width has increased more than the other two diameters. In fact, from the lateral roentgenogram of the skull alone this sella might have been considered "borderline."

When studying intrasellar tumors, it should be remembered that pituitary adenomas frequently have significant suprasellar extension. Therefore, to obtain a correct determination of the volume of adenomatous pituitary glands we cannot limit ourselves to the plain skull roentgenograms. Encephalography and in some cases bilateral carotid angiography may be necessary. In addition, in adenomatous pituitary glands, since they tend to take a more spheroid shape rather than the ellipsoid shape of the normal, the formula for the volumetric measurement of a spheroid rather than an ellipsoid should be used. These points, however, will be taken into

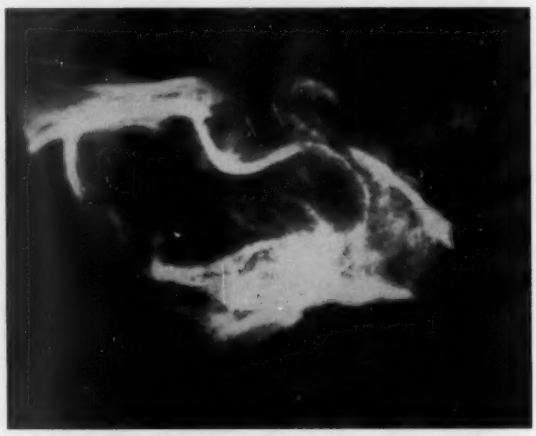


Fig. 18. The calcified diaphragma sellae connects the tuberculum sellae with the dorsum sellae.

detailed consideration in a future work now in process dealing with the sella in cases of intrasellar tumors.

SUMMARY

The width (third dimension) of the sella turcica may be obtained from the contour of the sellar floor recognizable in over 90 per cent of the cases on the standard posteroanterior roentgenogram of the skull. With the help of laminagraphy the sellar floor is demonstrated on the frontal roentgenograms in all cases. The sella, as any other anatomic structure, should always be studied three-dimensionally. Some of the advantages connected with the three-dimensional appraisal of the sella are indicated. When the sella enlarges, its width enlarges too. Sellas considered normal or even small may prove to be large when studied three-dimensionally because they have expanded only or mainly in one diameter; on the other hand sellas considered to be large on the lateral roentgenogram may prove to be within normal limits because their width is small. When the sella enlarges, it becomes almost invariably concave in its coronal outline, vaguely resembling a saucer. Anatomic evidence is offered that the width of the sella corresponds to the width of the pituitary gland. With three diameters of the sella being now available, it is possible to calculate the volume of the sella and with some limitations the volume of the pituitary gland. Such calculations have been carried out and verified with anatomic specimens in 10 cases. A roentgenographic determination of the volume of the pituitary gland *in vivo* is, with certain limitations, feasible. A volumetric appraisal of the size of the sella is more accurate than the linear and area methods used up to now. A very simple method of calculating the volume of the pituitary gland directly from the roentgenograms *in vivo* is suggested.

CONCLUSIONS

1. The width of the sella turcica has been roentgenographically reproduced.
2. The advantages of a three-dimensional study of the sella have been shown.
3. It has been proved that the width of the sella as determined by roentgenograms corresponds to the width of the pituitary gland.
4. A roentgenographic method of measuring the volume of the pituitary gland *in vivo* now appears feasible.

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This work would not have been possible without the collaboration of Mr. Joseph Morel, chief X-ray technician of the Diagnostic X-ray Department of the Clinical Center, National Institutes of Health. Dr. Frank Macri very carefully carried out the volume measurements of the pituitary specimens. Mrs. Doris Sadowsky is responsible for the mathematical and statistical calculations.

REFERENCES

1. Bloch, H., and Joplin, G. Some aspects of radiological anatomy of pituitary gland and its relationship to surrounding structures. *Brit. J. Radiol.*, 1959, *32*, 527–532.
2. Engels, E. P. Roentgenographic demonstration of hypophysial subarachnoid space. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *80*, 1001–1004.
3. Farinas, P. L. Value of x-ray examination of sella turcica in sagittal positions. *Radiology*, 1939, *32*, 411–415.
4. Hughson, W. Meningeal relations of hypophysis cerebri. *Anat. Rec.*, 1922, *23*, 21.
5. Mahmoud, M. E. The sella in health and disease. *Brit. J. Radiol.*, 1958, Suppl. 8, p. 9.
6. Oppenheimer, D. R. Some pathological findings in cases with radioactive pituitary implants. *J. Laryng. & Otol.*, 1959, *73*, 670–678.
7. Orley, A. Neuroradiology. Charles C Thomas, Publisher, Springfield, Ill., 1949, p. 169.
8. Rasmussen, A. T. Quantitative study of human hypophysis cerebri, or pituitary body. *Endocrinology*, 1924, *8*, 509–524.

SOME REMARKS ON ENCEPHALOGRAPHY*

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DURING the past decades neuroradiology has become more and more a part of routine examination procedures. It is especially so because of the Swedish investigators, Lysholm and later Lindgren[3,4] (and his pupils), who have fascinated us with their advances in the study of the contents of the bony skull by means of air insufflation, both as regards the examination technique as well as the interpretation of the roentgenographic images. A wealth of data has already been recorded because of painstaking and concentrated work on the part of individuals and groups. This work is still going on. Apart from Twining and Epstein,[2] the names of Dyke and Davidoff[1] should also be mentioned in this respect. They have described the normal and pathologic criteria so clearly that the cerebrum lies before us like an "open book."

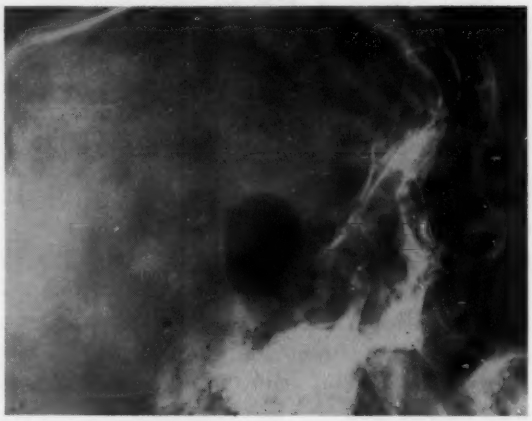

FIG. 1. Survey roentgenogram of the skull shows spontaneous air filling in a case of parasellar meningioma.

PROCEDURE

Preparation. The patient is examined in a fasting condition, preferably in the morning. Premedication is not necessary, but very nervous or agitated patients derive much benefit from luminal or some other barbituric acid derivative; epileptics are given an anti-epileptic agent. Preliminary survey roentgenograms of the skull (including the base) are indispensable, since one is often confronted with unsuspected, spontaneous pathologic air fillings (Fig. 1), calcifications or decalcifications, and/or asymmetries which may remain hidden. Also, the findings on the routine skull roentgenogram form a part of the total picture. The ear-flaps of the patient are folded forward and fastened with thin plastic adhesive tape during the roentgenographic examination to prevent misleading shadows in the area of the fourth ventricle and aqueduct. The lumbar puncture needle is inserted at the level of L 4–5 or L 3–4 with the patient lying on his side. After taking the cerebrospinal fluid pressure and performing the Queckenstedt test, the patient is placed in the sitting position with the body slightly bent forward and the head moderately flexed. Usually the head leans against the vertically tilted top of the Lysholm table, the chin being supported by means of a simple device. At this point the insufflation proper can be started. The cerebrospinal fluid obtained during the pressure determination ($\pm$1.0 ml.) is reserved for cytodiagnosis. No fluid is withdrawn in cases of posterior fossa tumors and coning (Fig. 2).

Method. The examination must be roentgenologically guided so that everything is visualized step by step, with full attention to details not only of the ventricles but also of the cisterns and sulci. An incomplete encephalographic examination gives an insufficient and incorrect understanding of the intracranial processes; a complete one makes it possible to observe even minute changes. The radiologist should not give

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an opinion until after he studies the ventricles, cisterns and sulci, particularly when he concludes that there is no evidence of abnormalities.

Knowledge of the normal appearance of the sulci and cisterns is, of course, necessary in order that the insidious onset and seemingly paradoxical progress of the lesions may be recognized. A space-occupying process need not cause displacement over the mid-line; the expansion may have taken place at the expense of sulci and cisterns. A septum pellucidum and a third ventricle situated in the mid-line, therefore, do not exclude by any means infiltrations and other processes.

It is wise to number the roentgenograms, to use high voltages with short exposure times, and to employ cassettes with a built-in Lysholm grid for the lateral views. The intensifying screens should be immaculate and moderately rapid. The Schönander table TIT5 with the Lysholm skull holder and a graded arc are of definite advantage.

Preference of Lumbar to Suboccipital Puncture. As stated by Lindgren[3,4] and Robertson,[5] a good lumbar injection technique affords a greater freedom of movement, not only to the physician and his staff but also to the patient. When the suboccipital puncture is used, the manipulations with cassettes and cassette holder always take place in the immediate neighborhood of the needle, thus increasing the chances of infection.

Fig. 2. Coning of the tonsils in a case of posterior fossa tumor.

Fig. 3. Filling of the tentorium cupula and subdural air over the clivus and sella turcica.

A further disadvantage of the suboccipital technique is the difficulty of executing the extension or rotatory movements in the event of unsuspectedly low tonsils and a flattened cisterna magna. This difficulty is especially marked in filling the pontocerebellar, interpeduncular and chiasmatic cisterns. On occasion, the lumbar needle is placed in the wrong position, in which case preliminary roentgenograms show the air subdurally (Fig. 3, 4, 5, and 7). A second puncture, one vertebra higher, will remedy this situation in a simple way.

Quantities of Cerebrospinal Fluid Withdrawn and Air Used. Even in the early days of encephalography, it was thought proper to withdraw as much cerebrospinal fluid as possible and to replace it by air, if need be with slight overpressure. In 1958, Schnitker and Ulrich[6] still removed 50 to 220 cc. of fluid (until the system was empty). I do not believe that this is wise as it leads to serious subjective complaints during the examination lasting up to some days afterwards, and I have also gained the strong

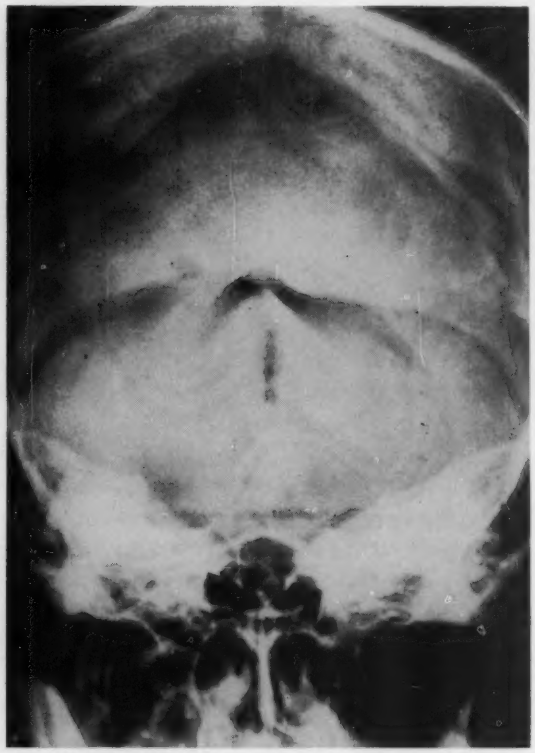

Fig. 4. Posteroanterior encephalogram shows air below the tentorium.

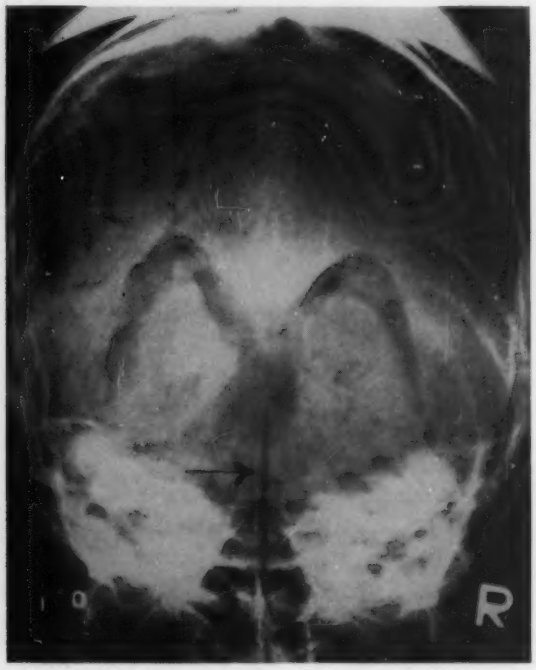

Fig. 5. Subdural air reaches the fissura longitudinalis.

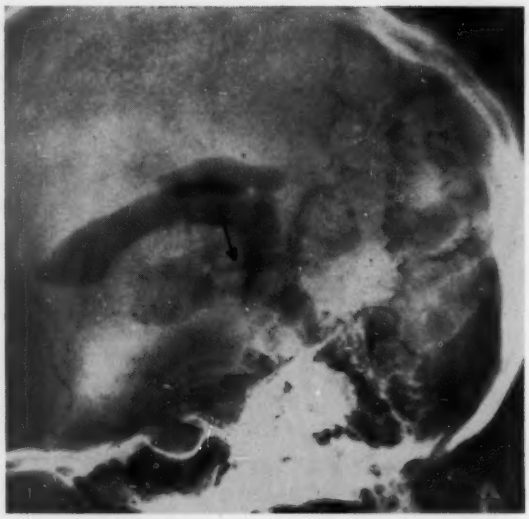

Fig. 6. Air in cisterna venae magnae, overlying the corpora quadrigemina.

impression that such encephalograms may be misleading as a result of technical errors. A remarkably high percentage of subdural air collections is reported on the first and second days of the examination. This is in sharp contrast to the occasional case of subdural air collection found on approximately 300 encephalograms made in the St. Ursula Clinic in the past fourteen months. I fully agree with Slosberg and others[7] that withdrawal of a minimum of cerebrospinal fluid with insufflation of a varying amount of air makes encephalogra-

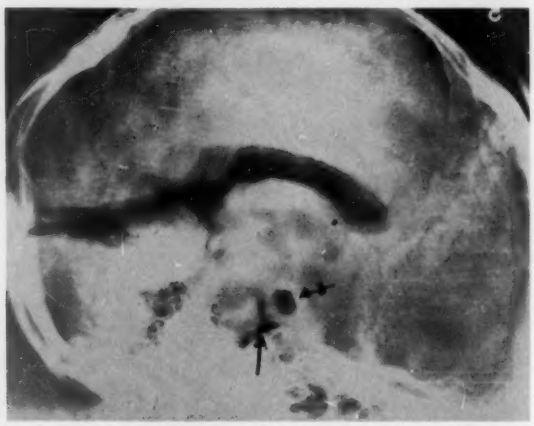

Fig. 7. Subtentorial air and subdural air over the clivus (arrow) and partial filling of the cisterna interpeduncularis (double arrow).

phy well tolerated. The patients have remarkably few complaints and there are much less autonomic reactions, in particular no troublesome vomiting and no profuse sweating. The headaches are also less severe.

Depending on the sizes of the fluid spaces, 30 to 60 cc. of air is instilled, and at the most one quarter of this volume of cerebrospinal fluid is removed, although the withdrawal of as little as 2 to 4 cc. is excellently tolerated. The patients cooperate actively in the examination, and twenty-four hours later, unlike those in whom much fluid has been removed, are quite cheerful. Opinions vary on the sequence of the procedure, although there is a tendency first to insufflate and then to withdraw the fluid for every fractional replacement. It is, as a rule, better to begin with insufflation, especially in view of the increasing possibilities of encephalography in the detection of processes in the posterior fossa. Only after the position of the tonsils is checked (Fig. 2) and the ventricles are filled with 15 to 25 cc. of air, is the cerebrospinal fluid slowly withdrawn in fractions up to maximally one fourth of the volume of air insufflated.

The ascent of the air is also facilitated by this technique, resulting in good filling of the crypts with the fluid.

Value of the Cerebrospinal Fluid Determinations. According to Robertson,[5] the number of cells increases during the insufflation. In 100 epileptics, however, no increased number of cells was demonstrated in the cerebrospinal fluid withdrawn after 20 to 25 cc. of air had been insufflated in fifteen to twenty minutes. Of late, we try to prevent the risk of obtaining too high a cell count by using only the first 1 to 1.5 cc. (obtained in the pressure measurement) for this determination.

Speed of Insufflation. According to Robertson,[5] this should be 1 cc. of air per minute. I agree that slow insufflation causes the least number of subjective complaints, but feel that a somewhat more rapid passage is still possible, with individual variations from 1 to 3 cc. of air per minute. Filling of the cisterns is always clearly indicated by complaints: filling of the cisternae ambientes causes pain in the temples; filling of the cisterna chiasmatis and frontal sulci gives rise to pain over the eyes, often attended by nausea.

After the ventricles are sufficiently filled, it is possible to effect good filling of the cisterns more rapidly without provoking more severe complaints than on slow filling. The filling is well tolerated and, so to speak, in a resigned manner in cases of cerebral and cortical atrophy. Tumors of the large cerebral hemispheres often cause a rapid and severe pain reaction and nausea. Filling in patients with processes of the posterior sulcus is usually well tolerated.

Room air is suctioned through a sterile gauze and then used for the insufflation. This has never led to complications. Although the use of oxygen is recommended by others because it is absorbed better and more rapidly, there is, in view of the good results obtained with the working method described above, no evident need to resort to oxygen. Moreover, the twenty-four hour encephalogram is of great importance. We, therefore, advise against oxygen breathing (in tent or by means of goggles) for some hours after the end of the examination. By then the slight quantity of air insufflated will be fully absorbed in the great number of cases.

The Fractional Insufflation Step by Step. After slow insufflation of 5 to 7 cc. of air with moderately flexed head, the air collects mainly in the cisterna magna. From this site the air will, so to speak, flow over through the foramen Magendie, which causes filling of the fourth ventricle, aqueduct of Sylvius and posterior part of the third ventricle. In the case of a small cisterna magna this happens a few seconds after the beginning of the insufflation. If, however, the cisterna magna is large and high, the cerebrospinal fluid level has first to fall to the level of the foramen Magendie and the filling of the ventricles will be delayed. One should, therefore, guard against

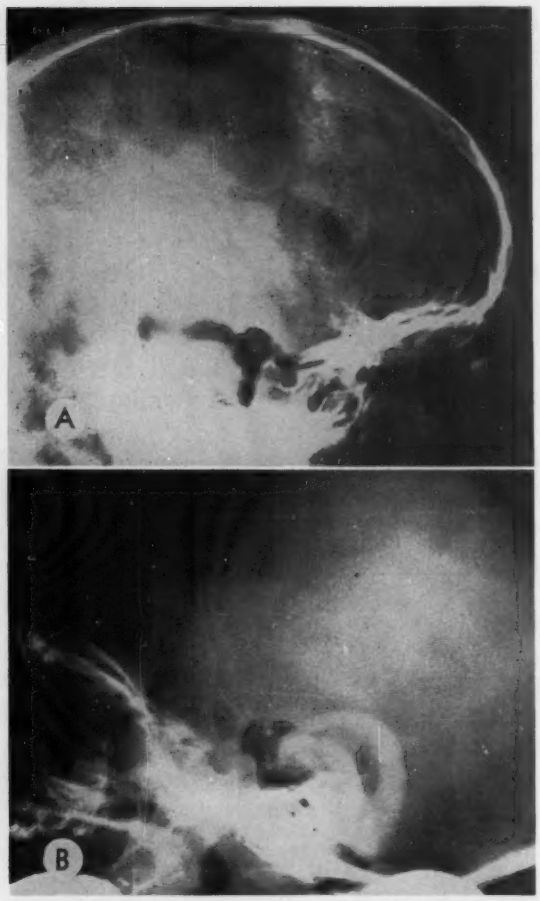

Fig. 8. (*A*) Complete filling of the cisterna interpeduncularis (same case as in Figure 7). Note the sharp concave border of the cisterna interpeduncularis as compared to the border of subdural air over the clivus. The lateral projection of the temporal horn is visible. (*B*) Filling of the cisterna interpeduncularis in a child three months old. Compare the distance from the clivus to the cisterna interpeduncularis with that in adults. The distance is not due to tumor. The shadow between the air filled cisterna and the osseous part of the clivus is cartilage bone.

bringing the head in extension if the first roentgenograms do not show filling of the fourth ventricle. The result would be a premature filling of the cisterna chiasmatis and frontal sulci, causing much discomfort to the patient. The posture of moderate flexion should be maintained and the (large) cisterna magna filled further. With slow insufflation and a proper head position, pure ventricular filling may occur; however, at this time the air often presents spontaneously in the cisterns of the posterior sulcus. Should this not occur or should it be incomplete—with satisfactory filling of the lateral ventricles—rapid instillation of 5 to 7 cc. of air with the head kept in a flexed position is recommended. If necessary, this may be repeated. The patient then feels a violent but short-lasting headache.

An attempt is made to obtain complete filling roentgenograms of the following cisterns: (1) cisterns at the upper side of the cerebellum; (2) cisterna venae magnae, cisterns adjoining the corpora quadrigemina at the upper side (Fig. 6); (3) cisterna ambiens on both sides; (4) cisterna interpeduncularis (Fig. 7 and 8, *A* and *B*) and cisterna cruris (Fig. 9) on both sides; (5) cisterna pontis; and (6) cisterna pontocerebellaris. The cisterna chiasmatis is also often filled, as well as a narrow cistern situated above the third ventricle.

ROENTGENOGRAPHIC TECHNIQUE

At the beginning, roentgenograms are made in two projections, at right angles. This not only facilitates the interpretation, but also enables us to make a reliable ana-

Fig. 9. Filling of the cisterna cruris. Compare with Figure 10.

lysis. This would otherwise be impossible.

The image of the third ventricle (in the beginning not more than an indication in the lateral and posteroanterior views) becomes gradually complete.

Only after a satisfactory filling of the cisterns and ventricles is the patient allowed to lie flat on his back. Before this, the ears are untaped and the patient remains sitting with bent head until everything is ready.

As soon as the patient lies down, some exposures are rapidly made with horizontal rays in order to evaluate the cisterna chiasmatis and anterior part of the third ventricle. With the patient on his back, the air, which had been trapped in the cisterna magna and cisterna pontis, ascends. This is usually more than sufficient for good visualization of the basal cisterns and the sulci. It sometimes proves advantageous to try to fill the anterior part of the third ventricle while the patient is in the sitting position by extending the head. This is best done when the cisterns are not yet filled. The anterior part of the third ventricle is also filled while the patient is lying down. If necessary, the head can be bent further backwards. This should be done slowly and with great care so as to avoid vomiting and nausea.

Fig. 10. Filling of the sulci olfactorii in a case of basichromophobe adenoma of the pituitary gland.

In the supine position, lateral, anteroposterior and semi-axial roentgenograms are made. The lateral view has been discussed above, the other two views give information regarding the anterior horns and cellae mediae of the lateral ventricles, the anterior and central part of the third ventricle, and the septum pellucidum.

The examination for visualization of the posterior horns and trigona takes place in a similar way, in the prone position. The posterior horns are often asymmetric (congenital).

I consider the directed examination of the temporal horn one of the essential parts of encephalography, since a destructive, infiltrative process can still be present in the region of a temporal horn even in a case where the septum pellucidum and the third ventricle are situated in the mid-line. On the other hand, displacements of the septum pellucidum and the third ventricle can be observed, while more detailed information about the location and the extent of a tumor can only be obtained by studying the form and position of the temporal horns. The encephalograms of the temporal horn provide an extra dimension by which to localize processes which are not always situated supratentorially. Large, slowly growing processes of the pontine angle or pons may cause slight changes in the position of the temporal horns.

Procedure of Filling the Temporal Horns. Both temporal horns can be filled with the forward somersault (Fig. 13), but unfortunately the lateral exposure must be omitted. It is best to follow the method of the Swedish school in which only one temporal horn is filled at a time.

In order to fill the right temporal horn, the patient is placed in the prone position. With the help of at least two men, he is laid so that his head is well over the edge of the

Fig. 11. Air over cerebellum. The air was correctly insufflated. Compare with Figure 12, where the air was placed subdurally.

Fig. 12. Subdural air over cerebellum. Compare with correctly placed air over cerebellum in Figure 11.

table. We take his head in our hands quite firmly and bend it so that the chin is towards the chest. Held fast in this way, the head is then slowly rotated 90 degrees so that the right side turns upwards. The chin remains against the chest. In the meantime, an aid has placed the patient on his left side. The head remains constantly lower than the neck and is kept in the lateral position for one minute. The right temporal horn now empties slowly; the cerebrospinal fluid disappears from the supracornual fissure and is replaced by air. Subsequently, the head is—again slowly—turned with the nose upwards, but it is still bent and lower than the body. When the position is reached in which the patient lies on the back of his head, the chin is raised slowly but the head still is not placed higher than the body. Then two encephalograms are made, approximately in the reversed "Tschebull position:" one in the longitudinal axis of the temporal horn and another in the lateral view. The position of the head is accurately measured and recorded with a graded arc with respect to the Frankfurter horizontal.

The left temporal horn is filled in a similar manner. I do not advocate the method in which the patient is placed upright in order to empty the right temporal horn which was previously filled, as this is attended by marked discomfort on the part of the patient.

After filling of the left temporal horn, encephalograms, in the anteroposterior and in the lateral view, are taken in identical positions so that a comparison of the two

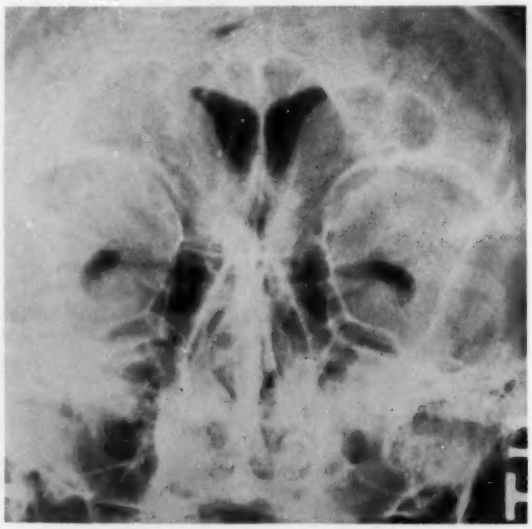

Fig. 13. Temporal horns filled at the same time by a forward somersault movement.

horns can be made. The angle made with the Frankfurter horizontal is not the same for the different skulls. Time should be taken to determine the correct angle so that the characteristic crescent or half-moon configuration can be visualized on the anteroposterior encephalogram. The correct projection is usually more closely approached with greater extension of the head.

The Sulci. The examination of the sulci is the final part of the encephalographic procedure. The depth of the sulci is important in determining the presence of cortical atrophies, local or generalized. This is especially so in cases of Alzheimer's disease, Pick's disease and in porencephalies. The fact that the sulci do not fill may indicate adhesions. Local malformations and abnormal (asymmetric) configurations may be indications of a tumor.

SIGNS OF DISCOMFORT

Attention should be paid to simple criteria as, for example, the color of the face. Pallor, cold nose and not rarely a slow and weak pulse indicate a threatening collapse. This should not be regarded as an alarming condition, and one should not hasten to withdraw the cerebrospinal fluid nor should the patient be made to sigh frequently and deeply. It is better to insufflate the air somewhat more rapidly. Further measures are to talk to the patient encouragingly and to have him or her smell something refreshing. Should the patient collapse in spite of mechanical support, he should be placed on his side. After the patient lies quietly for two to five minutes, it is usually possible to resume the examination and to finish it without further complications. Sometimes the recovery can be speeded up by an intramuscular injection of coramine.

DANGERS, INDICATIONS, CONTRAINDICATIONS AND ADVANTAGES

Provided the correct precautions are taken, encephalography is a harmless procedure. It is contraindicated in acute, active, recent vascular processes, especially in aneurysms of the circle of Willis. It is not improbable that the fatalities in cases described in the literature were caused by the examination itself rather than by the use of air which was incriminated as the noxious agent.

With the method described, good results are also obtained in processes of increased pressure and threatening herniation (Lindgren, Göster, Horle and others). This method is superior to ventriculography in that it clearly defines the anatomic relationships in the posterior sulci, unless, of course, the patient is unable to cooperate or is in a subcomatose state.

Extrapontine and intrapontine processes and lesions in the region of the pontine angle are especially suitable for detection by encephalography.

When there is a suspicion of diencephalic, sellar and parasellar tumors, one should not be tempted to fill the ventricles or cisterns alone. Only a complete encephalographic examination will enable the radiologist to supply the necessary preoperative information to the neurosurgeon. A study of a particular situation and the filling of the third ventricle and temporal horn, olfactory sulcus,. cisterna chiasmatis and cisterna interpeduncularis together may lead to accurate demarcation of tumors. Arteriography often may be indispensable in determining a possible richness in vascularization and unsuspected displacements of anterior and posterior cerebral arteries.

It is not impossible that an abnormality visualized on encephalograms may be caused by a large aneurysm.

ANATOMIC RELATIONS

A successful encephalogram is a cross section *in vivo*. The stem between the cisterna pontis (behind Blumenbach's clivus) and the fourth ventricle and aqueduct is visible. Over this, sometimes as a somewhat accentuated "whiteness," the peduncle projects itself behind the cisterna interpeduncularis. The rear side of the third ventricle borders on the pineal gland, under which a long air shadow, parallel to the

aqueduct, is seen—the cisterna venae magnae. Between this and the aqueduct, the corpora quadrigemina throw their projections. Halfway in the aqueduct a slight indentation is visible, corresponding to the space between the anterior and the posterior parts of the lamina quadrigemina. The cisternae ambientes embrace the peduncles; together they form a fork, in the lateral view, 5 to 11 mm. behind the posterior commissure. The pulvinar is situated between the wings of the cisterna ambiens and this cistern itself.

The anterior demarcation of the round cisterna interpeduncularis is often convex, sometimes sharply delimited by a membrane, sometimes separated a few millimeters from the cisterna chiasmatis situated before it. According to Schaeffer in 91 per cent of the cases the cisterna chiasmatis lies directly over the sellar diaphragm and in 4 per cent over and behind the dorsum of the sella turcica. The pontocerebellar cisterns are especially visible in the posteroanterior views along the petrous bone.

Filling defects here are only of significance if they persist after an effort is made to fill the cisterns concerned.

The olfactory sulci (Fig. 10) should not be confused with the cisternae cruris (Fig. 9). The first are situated more closely together and are longer; the latter are spread apart. The cisternae cruris are lateral extensions of the cisterna interpeduncularis.

The tentorium forms a cupula over the cerebellum, but a separate cisterna cerebelli (Fig. 6) can often be discerned underneath the latter (Fig. 11 and 12). The tuber cinereum and the mammillary bodies can be localized, using as a basis the demarcations of the third ventricle and the basal cisterns. A filling defect, the massa intermedia, is often present in the third ventricle. In the anteroposterior view the third ventricle presents as an ovoid shadow. Above it, between the anterior horns, the septum pellucidum is visualized which may be up to a few millimeters thick. The supracornual fissure of the temporal horn, which extends over the uncus and hippocampus, may be asymmetric, due to the difference in the development of the collateral eminentia.

It may be difficult to fill the cisternae cruris, but this may be effected by repeated insufflation with the head extended. The cisterna pontis is often hidden behind the mastoids. In this case a detail view with higher voltage is recommended. Generally, the cisternae pontocerebellares fill spontaneously on both sides; however, the filling may be retarded on one side. In this case then, the filling is accomplished with insufflation of 5 cc. of air with the head slightly bent and slanting somewhat toward the nonfilled side. The lateral views of the cisternae chiasmatis and interpeduncularis should always be supplemented with a semi-axial view. This is to prevent a unilateral filling from escaping our attention and to enable us to interpret disturbing secondary shadows (cisterna ambiens, temporal horn, fissure of Sylvius) correctly.

The Twenty-four Hour Encephalogram. Porencephalic cysts sometimes fill after twenty-four hours. Further, the twenty-four hour encephalogram indicates the speed of absorption. In some cases a dilatation which previously was obscure becomes defined. Occasionally, subdural fillings take place unexpectedly and sometimes the subarachnoidal sulci do not appear to be dilated until after twenty-four hours. Now and again the diagnosis of a convexity adhesion, which seemed to be correct in the beginning, had to be discarded because of further findings on the twenty-four hour encephalogram.

SUMMARY

A description of an encephalographic technique based on the Swedish method is given along with some practical suggestions and criteria. Encephalography, performed by the radiologist himself, should give a complete picture of the cisterns, sulci and ventricular system.

Some typical encephalograms are shown.

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REFERENCES

1. Davidoff, L. M., and Dyke, C. G. The Normal Encephalogram. Second edition. Lea & Febiger, Philadelphia, 1946.
2. Davidoff, L. M., and Epstein, B. S. The Abnormal Pneumoencephalogram. Second edition. Lea & Febiger, Philadelphia, 1955.
3. Lindgren, E. Some aspects of technique of encephalography. *Acta radiol.*, 1949, *31*, 161–177.
4. Lindgren, E. Radiological examination of brain and spinal cord. *Acta radiol.*, 1957, Suppl. 151.
5. Robertson, E. G. Pneumoencephalography. Charles C Thomas, Publisher, Springfield, Ill., 1957.
6. Schnitker, M. T., and Ulrich, R. P. Observations on twenty-four hour pneumoencephalogram with special reference to diagnosis of cortical atrophy. *Radiology*, 1958, *70*, 15–22.
7. Slosberg, P., Bornstein, M., and Lichtenstein, R. Pneumoencephalography with minimal withdrawal of cerebrospinal fluid. *J. Mt. Sinai Hosp. New York*, 1955, *21*, 299–305.

EMULSIFIED IODIZED OIL VENTRICULOGRAPHY*

By TOSHIO MATSUBARA, M.D., *and* TAKAYOSHI NOMURA, M.D.
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ROENTGENOGRAPHIC examination of the cerebral ventricular system is an important adjunct in neurosurgical diagnosis. Up to the present, chiefly pneumoventriculography and lipiodol ventriculography have been employed for this purpose. The contrast procedures, however, have failed to completely satisfy the needs of neurosurgeons, particularly in the visualization of the third and fourth ventricles and of the sylvian aqueduct. The disadvantage of the usual contrast media used (air and iodized oil) is that they are not mixable with water, *i.e.*, with cerebrospinal fluid. Unless the contrast substance can spread through the ventricular space, independent of external factors, the result will vary with the technique employed and the diagnosis therefore may not always be correct. The ideal contrast medium in ventriculography must be diffusible in the cerebrospinal fluid.

After much experimentation, we have prepared an emulsion of iodized oil for the purpose of obtaining a more satisfactory visualization of the intracranial spaces.[1,2,3]

PREPARATION OF THE EMULSIFIED IODIZED OIL

(A) A neutral isotonic solution of sodium lauryl sulfate is prepared by dissolving 4 gm. of sodium lauryl sulfate in 100 ml. of water and by adjusting the pH to neutral with 0.5 per cent (isotonic) hydrochloric acid using methyl red as an indicator. This solution is sterilized and diluted 4 times with artificial cerebrospinal fluid[3] or with Ringer's solution.

(B) The iodized oil used is a molecular uniform isobaric lipiodol (11.63 per cent iodine content†), the specific gravity of which is 1.005–1.007 at body temperature (37° C.) and similar to that of normal cerebrospinal fluid. It is not desirable to mix an oil of higher specific gravity with one of lower specific gravity to adjust the specific gravity of the iodized oil because, when such a mixture is emulsified, the two components of different specific gravity become separated again, and the emulsion particles are no longer stable in the cerebrospinal fluid.

(C) Next, 4 portions of isobaric iodized oil and 1 portion of a neutral isotonic solution of sodium lauryl sulfate are mixed and vigorously shaken, to produce emulsification. The ready emulsion is milk-like and is stable for at least several months at room temperature. Old emulsions which are unsuitable for use can be re-emulsified by severe shaking. When this emulsion is added to the cerebrospinal fluid, it spreads evenly and the emulsion particles remain stable for a few hours. Heating of the emulsion at higher temperature and for longer periods decreases the diffusibility of the emulsion particles.

DIRECTIONS FOR THE VENTRICULOGRAPHIC APPLICATION OF EMULSIFIED IODIZED OIL, ESPECIALLY FOR VISUALIZATION OF THE THIRD AND FOURTH VENTRICLES AND OF THE SYLVIAN AQUEDUCT

1. Shortly before use, the emulsified iodized oil must be shaken for a few minutes.

2. Unilateral ventricular puncture is made into the anterior horn of the lateral ventricle, with the tip of the needle directed at the foramen of Monro.

3. Using the above method, 10–20 ml. of cerebrospinal fluid is removed and replaced by the same amount of the emulsified iodized oil, which is injected slowly into the ventricular space. Soon after injection of the contrast substance (within several minutes)

† This was prepared by the "Daiichi Seiyaku" Co. Ltd., Tokyo, Japan.

* From the First Department of Surgery, Nagoya University School of Medicine, Nagoya, Japan.

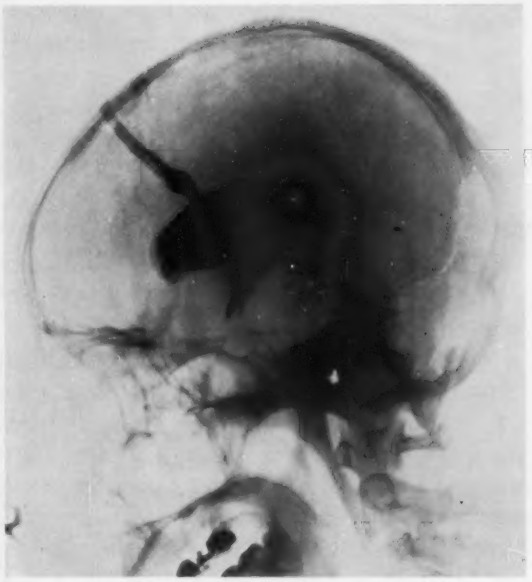

Fig. 1. Case 1. Lateral view of emulsified iodized oil ventriculogram. Normal.

Fig. 2. Case 3. Lateral view demonstrating an anterior third ventricle craniopharyngioma. The irregularities of the tumor surface can be seen.

ventriculograms are taken from various directions, without removing the puncture needle. Screening or changing the head position is not necesssry.

4. When the desired information has been obtained, the contrast substance is removed by irrigation of the cerebrospinal spaces with artificial cerebrospinal fluid or with Ringer's solution at body temperature until the cerebrospinal fluid becomes clear.

CLINICAL APPLICATION OF EMULSIFIED IODIZED OIL VENTRICULOGRAPHY

Emulsified iodized oil has been used in ventriculographies performed on 14 neurosurgical patients in order to visualize the ventricular spaces. The results are summarized in Table 1. Figures 1, 2 and 3 are ventriculograms of 3 of the patients.

SUMMARY

The preparation and clinical application of emulsified iodized oil in cerebral ventriculography are described.

We believe that emulsified iodized oil ventriculography has the following advantages: 1. Lipiodol suspension enables the whole ventricular system to be visualized on the ventriculogram since it spreads diffusely, not droplet-wise, into the cere-

Fig. 3. Case 12. Lateral view demonstrating the dilated lateral and third ventricles and the sylvian aqueduct with abnormal curvature and obstruction of the ventricular system at the inferior fourth ventricle.

TABLE I

RESULTS OF EMULSIFIED IODIZED OIL VENTRICULOGRAPHY IN 14 NEUROSURGICAL PATIENTS

Case No.	Patient Age Sex	Diagnosis	Ventriculographic Findings	Side Reactions		
				Temporary Fever (hours after examination, maximum elevation, duration)	Vomiting	Headache
1	Y.O. 20, M	Intraventricular tumor suspect	Normal	7 hr., 39.5°C., 1 da.	None	None
2	M.S. 12, M	Posterior fossa tumor suspect	Normal	6 hr., 39°C., ½ da.	Frequently during operation	Slight for 2 da.
3	N.T. 29, M	Craniopharyngioma in third ventricle	Third ventricle filled with a tumor the size of a hen's egg. Irregularity of the tumor surface also demonstrated	6 hr., 38.5°C., ½ da.	Twice during operation	None
4	K.U. 4, M	Pineal tumor suspect	Defect of posterior third ventricle	6 hr., 39.8°C., ½ da.	Twice during operation	None
5	T.E. 32, M	Intracranial metastasis	Communication of cyst with lateral ventricle		None	None
6	K.H. 12, M	Medulloblastoma	Defect of posterior third ventricle			
7	M.N. 44, M	Craniopharyngioma in third ventricle	Block of foramen of Monro		None	None
8	K.T. 15, F	Localized meningoencephalitis?	Shift of lateral ventricle		None	None
9	H.Y. 29, M	Acoustic neurinoma	Obstruction of aqueduct, symmetric dilatation of lateral and third ventricle	5 hr., 39°C., ½ da.	None	Slight for 1 da.
10	R.I. 26, M	Fourth ventricle tumor	Obstruction of aqueduct, symmetric dilatation of lateral and third ventricle	5–6 hr., 38.6°C., 1 da.		
11	E.I. 26, F	Posterior fossa tumor	Obstruction of aqueduct, symmetric dilatation of lateral and third ventricle	24 hr., 38.6°C., 1 da.		
12	C.K. 20, F	Acoustic neurinoma	Abnormal curvature of aqueduct	6 hr., 38.9°C., ½ da.		Slight for 2 da.
13	M.I. 35, M	Third ventricle tumor	Block of foramen of Monro			Slight for 3–4 da.*
14	S.K. 53, F	Brain stem tumor	Abnormal curvature of aqueduct			

* Continuous ventricular drainage was performed for 3 postoperative days.

brospinal fluid. 2. The narrow part, the third and fourth ventricles, and the sylvian aqueduct, can be demonstrated without troublesome procedures such as screening and changing the patient's head position. 3. After the ventriculograms are taken, the contrast substance can be removed by irrigation of the spaces with artificial cerebrospinal fluid or Ringer's solution, warmed to body temperature.

Side reactions following emulsified iodized oil ventriculography were temporary fever, which appeared after several hours, slight headache and, infrequently, nausea and vomiting, which were relieved without any medication. Such side reactions are believed inevitable in the process of a ventricular attack. When, however, an obstruction of the ventricular system is detected by ventriculography, it is desirable to carry out a continuous ventricular drainage for a few days and caution must be taken to prevent postventriculographic emergencies.

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REFERENCES

1. Matsubara, T. Water diffusible lipiodol as contrast medium for x-ray examinations of intracranial spaces (Part I). *Nagoya J. Med. Sci.*, 1956, *18*, 252.
2. Matsubara, T., and Ogawara, S. Water diffusible lipiodol as contrast medium for x-ray examinations of intracranial spaces (Part II). *Nagoya J. Med. Sci.*, 1957, *19*, 206.
3. Matsubara, T., and Nomura, T. Water diffussible lipiodol as contast medium for x-ray examinations of intracranial spaces (Part III). *Nagoya J. Med. Sci.*, 1958, *20*, 289.
4. Lewis, R. C., and Elliott, K. A. C. Clinical uses of artificial cerebrospinal fluid. *J. Neurosurg.*, 1950, *7*, 256–260.

THE ROENTGEN DIAGNOSIS OF INTRACRANIAL INCISURAL SPACE OCCUPYING LESIONS*

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MASSES occurring in the region of the incisura of the tentorium are usually extracerebral in origin. As such, the majority can be removed surgically. Unfortunately, the surgical approach is difficult and fraught with danger due to the possibility of injury to vital areas of the brain. Any attempt to remove these lesions surgically must be based on a very accurate preoperative anatomic diagnosis. The lesion must be evaluated as to size, exact location in relation to the brain stem, the edge of the tentorium, the middle fossa and the major arterial trunks. This can usually be accomplished roentgenologically. It is the purpose of this communication to present a group of lesions of the incisura and to analyze the roentgenologic findings.

ANATOMIC CONSIDERATIONS

The tentorial incisura is a rigid, roughly oval opening, circumscribed anteriorly by the dorsum sellae, laterally and posteriorly by the edge of the tentorium (Fig. 1). The upper portion of the brain stem (mid-brain) occupies most of the available space. In addition to the mid-brain the incisura contains the upper portion of the basilar artery with its bifurcation as well as the two posterior cerebral and superior cere-

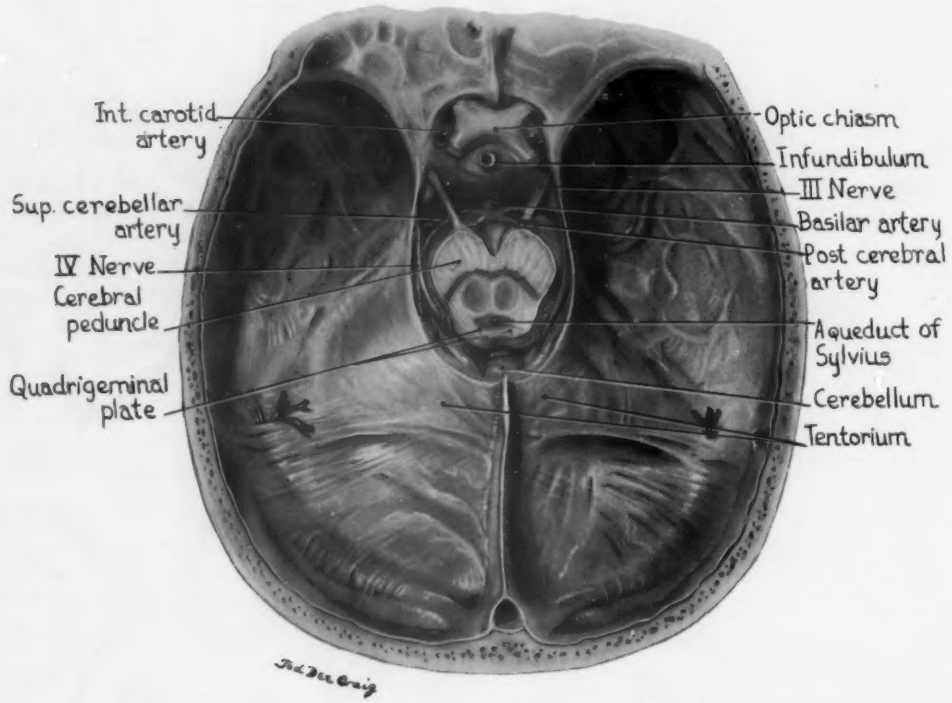

FIG. 1. The diagram represents the incisura of the tentorium. The mid-brain was sectioned transversely and the hemispheres have been removed.

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bral arteries (Fig. 1). The third and fourth cranial nerves are in the incisura, and the fifth and sixth nerves are just below it (Fig. 1). The posterior cerebral and superior cerebellar arteries are in the incisura as they turn around the brain stem. The posterior cerebral arteries leave the incisura as they continue their backward course towards the inferior and medial surfaces of the occipital lobes. The superior cerebellar arteries on the other hand go under the tentorium after turning around the mid-brain.

The structures contained with and adjacent to the tentorial incisura are extremely important. A glance at Figures 1 and 2 will show the close relationship of the tentorial edge with the cerebral peduncles, the quadrigeminal plate, the splenium of the corpus callosum and the vein of Galen as well as with the arterial trunks and nerves mentioned above.

Immediately above the incisura on each side is the medial side of the temporal lobe. In the center anteriorly is the hypothalamus and third ventricle.

CLASSIFICATION OF LESIONS

From the foregoing may be deducted that the lesions most apt to occur in this region would arise from (1) the mid-brain, (2) the edge of the tentorium, (3) the basilar artery or its branches, (4) the nerves, (5) the dorsum sellae and adjacent portion of the clivus, (6) the petrous tips, (7) the pineal body, (8) the vein of Galen.

These lesions would therefore be (a) gliomas of the mid-brain, (b) meningiomas arising from the tentorial edge or from the meninges over the upper portion of the clivus or petrous tips, (c) aneurysms of the basilar or of the posterior cerebral arteries, (d) neurinomas of the fifth nerve (neurinomas of the third and fourth nerves as isolated tumors are very rare), (e) chordomas, (f) pinealomas or pineal teratomas. Strictly speaking transtentorial herniations of the uncus and of the hippocampal gyrus are incisural masses. The same applies to upward herniations of the cerebellum. They

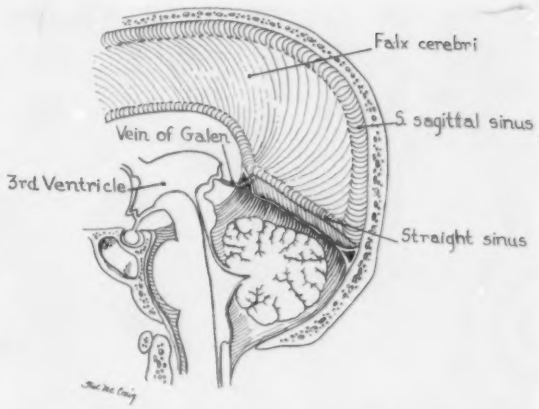

Fig. 2. Diagrammatic representation of the edge of the tentorium as seen from its lateral aspect. The brain stem, cerebellum and the third ventricle have been drawn in to illustrate the relative relationship of these structures to the tentorial incisura in the lateral projection. The vein of Galen is also illustrated but the basilar artery and its branches are omitted.

are not considered to be within the scope of this paper and will be mentioned only in the differential diagnosis.

The intrinsic gliomas of the brain stem are not strictly localized to the incisura but infiltrate upwards towards the thalamus and downwards towards the pons. They will not be discussed here. Pinealomas tend to grow towards the third ventricle and produce early obstruction of the aqueduct of Sylvius. Only occasionally do they grow chiefly posteriorly, towards the posterior portion of the cisterna ambiens. They will not be specifically discussed.

We are chiefly concerned with the extra-axial group of masses which occupy the region of the incisura. The most common of these, in our series of cases, have been the meningiomas. The second most common are the aneurysms of the bifurcation of the basilar artery and of the posterior cerebral arteries.

The material selected for discussion consists of 24 cases, of which 16 are meningiomas, 5 are aneurysms, and 3 are chordomas of the upper portion of the clivus.

ROENTGENOLOGIC FEATURES

In order to facilitate the description of the roentgenologic findings, the lesions will

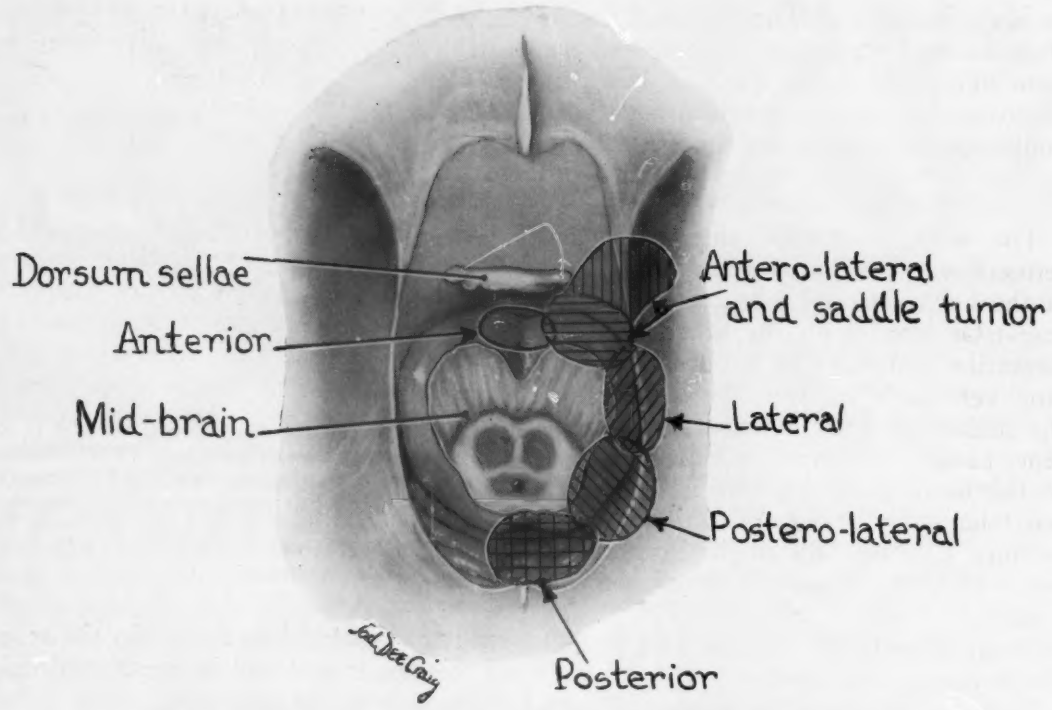

FIG. 3. Schematic representation of the relative location of the masses encountered in the region of the incisura in relation to the mid-brain.

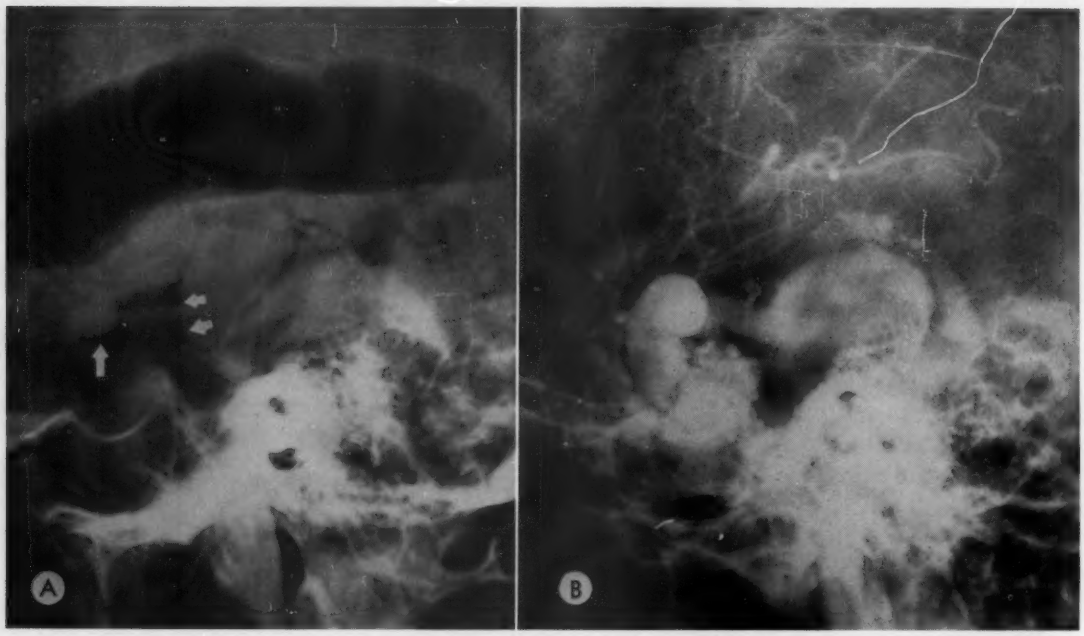

FIG. 4. *Arteriovenous malformation of the medial portion of the temporal lobe with large midline saccular dilatation of draining venous channels.* The patient (unit no. 73-83-51) was a forty-seven year old man who complained of spastic weakness of the right arm for a period of eight years. The pneumoencephalogram (*A*) reveals the presence of a mass encroaching on the basal cisterns at the junction of the cisterna pontis and interpeduncularis. These cisterns might be considered to be larger than normal. There is a smaller, rounded shadow in the suprasellar region (arrow). A carotid angiogram (*B*) demonstrates the arteriovenous malformation with very large saccular dilatation in the midline produced by dilated draining veins.

be divided into five groups depending on their location in relation to the brain stem. The groups are: midline anterior, anterolateral, lateral, posterolateral, and midline posterior masses (Fig. 3).

I. MIDLINE ANTERIOR MASSES

The midline anterior masses in our series of cases have been mostly aneurysms of the basilar artery at its bifurcation (3 cases). One case was an arteriovenous malformation of the temporal lobe with a very large venous sac in the midline anterior to the mid-brain (Fig. 4, *A* and *B*). Several other cases of aneurysm of the bifurcation of the basilar artery have been encountered but they are not large enough to produce a mass and are not discussed here. Only one meningioma could be classified as strictly midline in location. Epidermoid tumors may also occur here. We have encountered none that could be classified as incisural in location; they have been either retrosuprasellar or prepontine masses. One case (Fig. 5) thought to be an aneurysm of the basilar artery has not been confirmed by angiography and could possibly be an epidermoid tumor.

The plain roentgenograms may show bone destruction such as in chordoma (Fig. 6*A*), or sclerotic changes in the clivus and dorsum sellae such as in meningioma. Calcium deposits may be present either in meningioma or in chordoma. An aneurysm of the basilar artery may present shell-like calcification in its wall.

On pneumoencephalography the midline anterior incisural masses produce a deformity of the basal cisterns where the cisterna pontis and interpeduncularis come together. A segment of a round mass may be plainly visible (Fig. 4, *A* and *B*; 5; and 6, *A* and *B*). It is not uncommon to find enlargement of the cisterns around the mass due to atrophy of the adjacent brain, particularly in the vascular lesions (Fig. 4, *A* and *B*; and 5). Depending upon the size of the mass, the aqueduct may or may not be displaced posteriorly. In the frontal projection the aqueduct is in the midline.

The third ventricle shows either an increase in the concavity or true elevation of its posteroinferior aspect (Fig. 5). In the frontal projection it reveals no deformities or it may show a decrease in the height of its posterior portion.

Fig. 5. *Probable aneurysm of the basilar artery* (not proved by angiography as yet). The patient was a sixty-three year old man (unit no. 25-43-19) who complained of episodes of falling and unconsciousness for about a year and a half. The neurologic examination was essentially negative. The pneumoencephalogram demonstrates the presence of a rounded mass with a forward convexity encroaching upon the junction of the cisterna pontis and interpeduncularis. There is elevation of the posterior portion of the floor of the third ventricle (arrow). In spite of the large size of the mass, the aqueduct is not displaced dorsally. This might be due to secondary atrophy of the mid-brain and the upper portion of the pons.

Angiography is diagnostic in the vascular lesions (Fig. 4, *A* and *B*). In the chordomas and meningiomas vertebral angiography reveals posterior displacement of the superior aspect of the basilar artery (Fig. 11*B*).

To differentiate between the various types of midline anterior incisural masses, vertebral angiography is necessary. If there is bone destruction in the dorsum sellae and adjacent clivus, chordoma is the most likely possibility (Fig. 6, *A* and *B*). Meningiomas may produce sclerotic

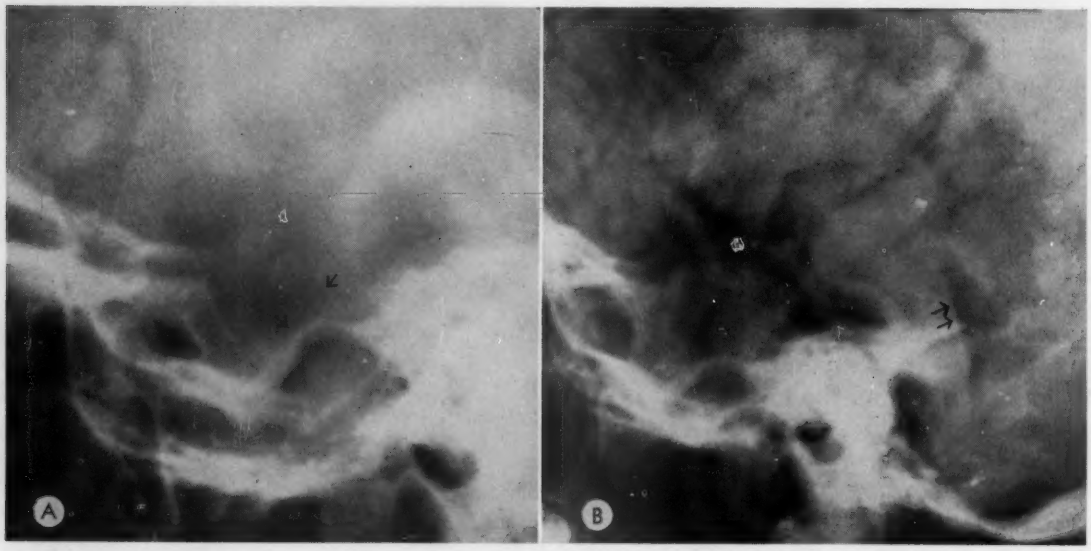

Fig. 6. *Chordoma involving the upper portion of the clivus.* The patient, a seven year old girl (unit no. 137-51-96), came in because of double vision of three months' duration. Examination disclosed a sixth nerve palsy on the left, facial weakness and diminished auditory acuity. The plain roentgenogram (*A*) shows destruction of the upper portion of the clivus and the posterior portion of the floor of the sella turcica (arrows). The pneumoencephalogram (*B*) reveals the presence of a rounded mass encroaching upon the basal cisterns at the junction of the cisterna pontis and interpeduncularis and extending slightly rostral to this point. The shadow of the basilar artery can be visualized and it is displaced backwards (arrow). The fourth ventricle (two arrows) shows a sharp anterior margin and is displaced dorsally.

changes in the bones of varying degree, occasionally actual bone destruction, or they may produce no visible changes. Calcium deposits with bone destruction indicate chordoma. Calcium deposits without bone changes may be due to meningioma.

II. ANTEROLATERAL MASSES

This group must be divided into two subgroups: (a) the solitary anterolateral masses, and (b) the saddle (or dumbbell) tumors.

(a) *Solitary anterolateral masses.* This subgroup was mostly composed of meningiomas attached to the free edge of the tentorium or to the tip of the petrous portion of the temporal bone (5 cases). One case was a blood clot produced by a bleeding aneurysm of the posterior communicating artery (Fig. 10, *A* and *B*). The remaining 2 cases were neurinomas probably arising from the fifth nerve. The cerebellopontine tumors are not included in this group because they are more lateral in location and are not centered as high as the truly incisural masses. Large cerebellopontine tumors, however, may extend through the incisura but this is a secondary defect.

The solitary meningiomas did not show any abnormalities on plain roentgenograms owing to their attachment to the free edge of the tentorium. None of the cases in this subgroup presented calcium deposits. There were no signs of long standing increased intracranial pressure except in one case where there was thinning of the dorsum and floor of the sella and increased prominence of the occipital emissary veins. The case of neurinoma of the fifth nerve presented erosion of the petrous tip without involvement of the internal acoustic meatus.

The characteristic roentgenologic findings on pneumoencephalography are (1) the lateral displacement of the aqueduct, and (2) the indentation on the posteroinferior aspect of the third ventricle (Fig. 7, *A* and *B*; and 8, *A* and *B*). To be diagnostic the maximum lateral displacement should be centered on the aqueduct; the fourth ventricle is displaced less than the aqueduct (Fig. 8, *A* and *B*). In most cases in this subgroup the aqueduct was not significantly dis-

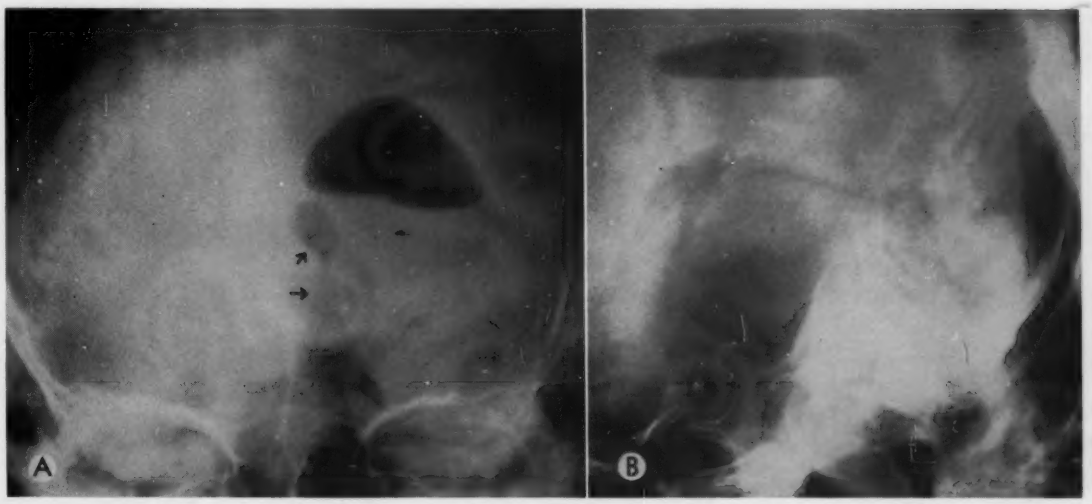

Fig. 7. *Neurinoma probably arising from the fifth nerve.* The patient was a fifty year old man (unit no. 142-41-73) who complained of stumbling for about three months with weakness particularly of the left knee and slight slurring of speech and difficulty in swallowing. The plain roentgenograms of the skull were normal. A pneumoencephalogram (*A*) reveals the presence of moderate lateral displacement of the aqueduct and upper portion of the fourth ventricle (lower arrow). There is an indentation on the posteroinferior portion of the third ventricle (upper arrow). The lateral view (*B*) shows only slight posterior displacement of the aqueduct and fourth ventricle. The tumor was found to be situated high in the cerebellopontine angle and to protrude through the incisura. The exact origin was not determined but it was found not to arise from either the seventh, eighth, ninth or tenth cranial nerve.

placed dorsad, but if the tumor is large some degree of dorsal displacement is usually present. A moderate degree of ventricular dilatation was present in all cases due to long standing partial obstruction, but air injected by the lumbar route entered the

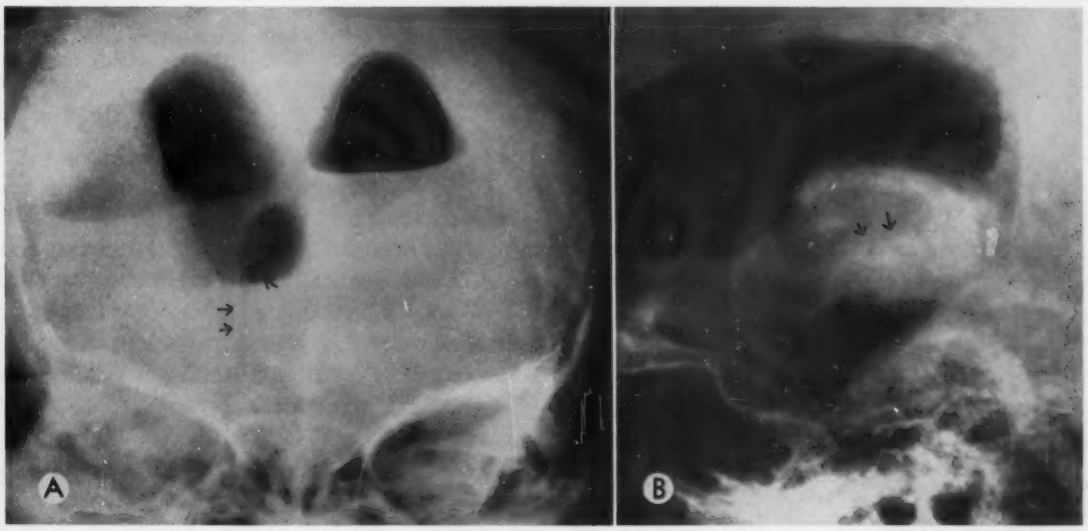

Fig. 8. *Meningioma arising from the edge of the tentorium.* The patient (unit no. 127-28-67) was a fifty-eight year old woman who complained of episodes of sudden weakness of both lower extremities which made her fall to the floor. She had had Ménière's syndrome for two years. The neurologic examination was noncontributory. She had a right Babinski sign. The pneumoencephalogram (*A*) reveals the presence of sharp lateral displacement of the aqueduct and upper portion of the fourth ventricle (two arrows). The lower portion of the fourth ventricle is at a nearly normal position. The entrance of the aqueduct is eccentrically placed in relation to the posterior third ventricle (upper arrow). The lateral view (*B*) shows moderate elevation of the posterior portion of the third ventricle (arrows)

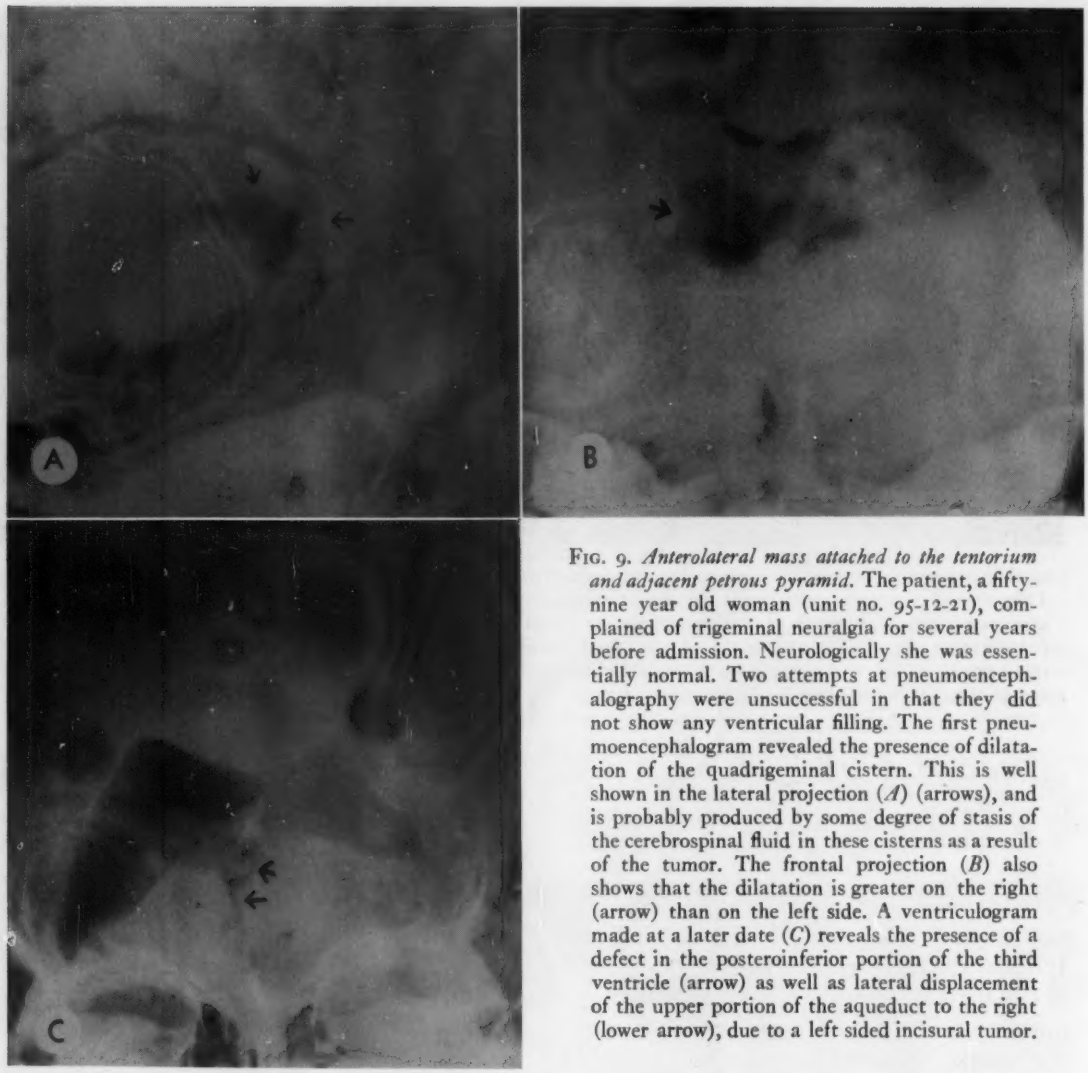

FIG. 9. *Anterolateral mass attached to the tentorium and adjacent petrous pyramid.* The patient, a fifty-nine year old woman (unit no. 95-12-21), complained of trigeminal neuralgia for several years before admission. Neurologically she was essentially normal. Two attempts at pneumoencephalography were unsuccessful in that they did not show any ventricular filling. The first pneumoencephalogram revealed the presence of dilatation of the quadrigeminal cistern. This is well shown in the lateral projection (*A*) (arrows), and is probably produced by some degree of stasis of the cerebrospinal fluid in these cisterns as a result of the tumor. The frontal projection (*B*) also shows that the dilatation is greater on the right (arrow) than on the left side. A ventriculogram made at a later date (*C*) reveals the presence of a defect in the posteroinferior portion of the third ventricle (arrow) as well as lateral displacement of the upper portion of the aqueduct to the right (lower arrow), due to a left sided incisural tumor.

ventricles easily except in one case which required ventriculography. In this latter case two attempts at pneumoencephalography were unsuccessful. They did reveal, however, a marked enlargement of the cistern over the quadrigeminal plate which was interpreted as secondary to interference with the circulation of cerebrospinal fluid in the posterior fossa with consequent formation of "fluid pools" in certain cisterns (Fig. 9, *A–C*; and 14, *A–D*). One of the cases of probable fifth nerve neurinoma also revealed a dilated segment of the cisterna ambiens.

Angiography was not performed in any of the meningioma cases in this subgroup. It is to be expected that vertebral angiography would demonstrate dorsal displacement of the basilar artery as in Figure 11*B* due to posterior displacement and rotation of the brain stem. The posterior cerebral artery would be displaced dorsally, medially and/or upwards. The superior cerebellar artery is displaced medially and dorsally.

In one instance of bleeding aneurysm arising at the junction of the posterior communicating and the internal carotid artery, a blood clot produced the angiographic signs of an incisural mass. There was up-

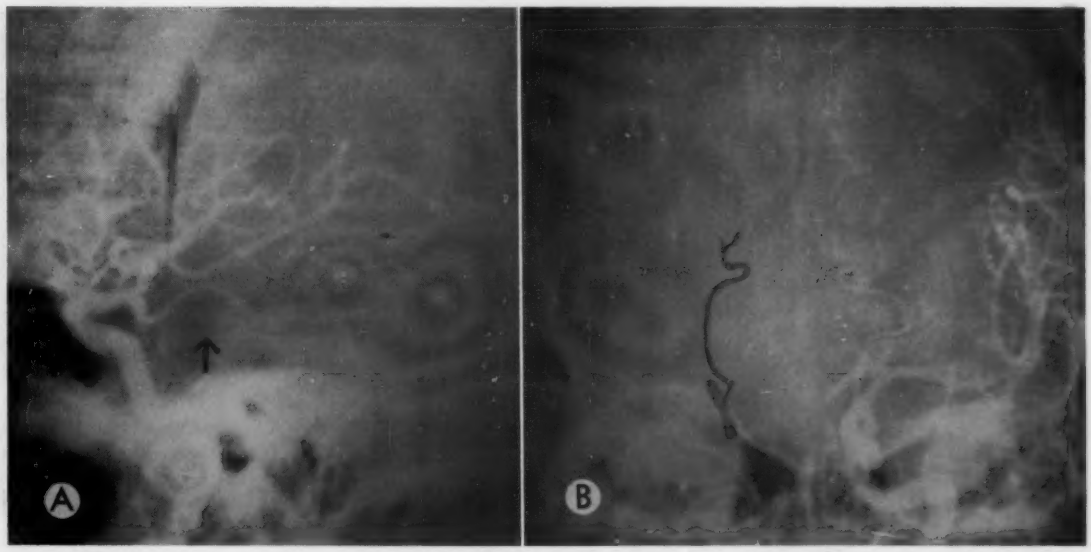

Fig. 10. *Bleeding aneurysm of the internal carotid artery at the junction with the posterior communicating artery, with blood clot.* The patient was a forty-nine year old woman (unit no. 141-50-26) who had a typical syndrome of a bleeding aneurysm upon admission. The lateral angiogram (*A*) shows elevation of the posterior communicating artery and the proximal portion of the posterior cerebral artery (arrow). There appears to be a carotid origin of the posterior cerebral artery on this side. The frontal angiogram (*B*) shows medial and upward displacement of the posterior communicating artery and of the proximal portion of the posterior cerebral artery. Compare with expected normal position sketched on the opposite side.

ward and medial displacement of the posterior communicating and proximal portion of the posterior cerebral arteries (Fig. 10, *A* and *B*).

(b) *Saddle (dumbbell) tumors.* There were 5 cases in this subgroup, all meningiomas. Jefferson[3] described 8 cases of neurinomas of the fifth nerve with a mass present in the middle and posterior fossae in at least 4 of the cases. Cushing and Eisenhardt,[2] and Castellano and Ruggiero[1] reported meningiomas of this type. They may be attached to the dura over the cavum meckelii or to the petrous pyramid near the apex (gasserian-petrosal meningiomas).

The plain roentgenograms demonstrated, in 2 cases, a peculiar sclerosis of the dorsum sellae and adjacent clivus, as well as of the medial portion of the middle fossa (Fig. 12, *A–C*). One case revealed partial destruction of the middle fossa and tip of the petrous pyramid which would be difficult to differentiate from a neurinoma of the fifth nerve (Fig. 14*C*). Two of the cases presented speckled calcification.

Pneumoencephalography was carried out in 3 cases and in one there was no ventricular filling. Vertebral angiography was then performed. In the other 2 cases ventriculography was carried out. In addition to the typical indentation on the posteroinferior portion of the third ventricle and the posterior and lateral displacement of the aqueduct, these cases present elevation of the temporal horn (Fig. 12, *A–C*; and 14, *A–D*). The indentation on the floor of the third ventricle may extend to involve its anterior portion (Fig. 13, *A* and *B*).

Vertebral angiography performed in 2 cases demonstrated dorsal displacement of the basilar artery (Fig. 11, *A* and *B*). Carotid angiography, in 2 other cases, disclosed elevation of the horizontal portion of the middle cerebral artery (Fig. 14*D*), as could have been predicted.

III. LATERAL MASSES

These masses are strictly lateral in location without an anterior or posterior extension. There was only one case that would fit this category, a meningioma arising from the lateral aspect of the tentorial edge.

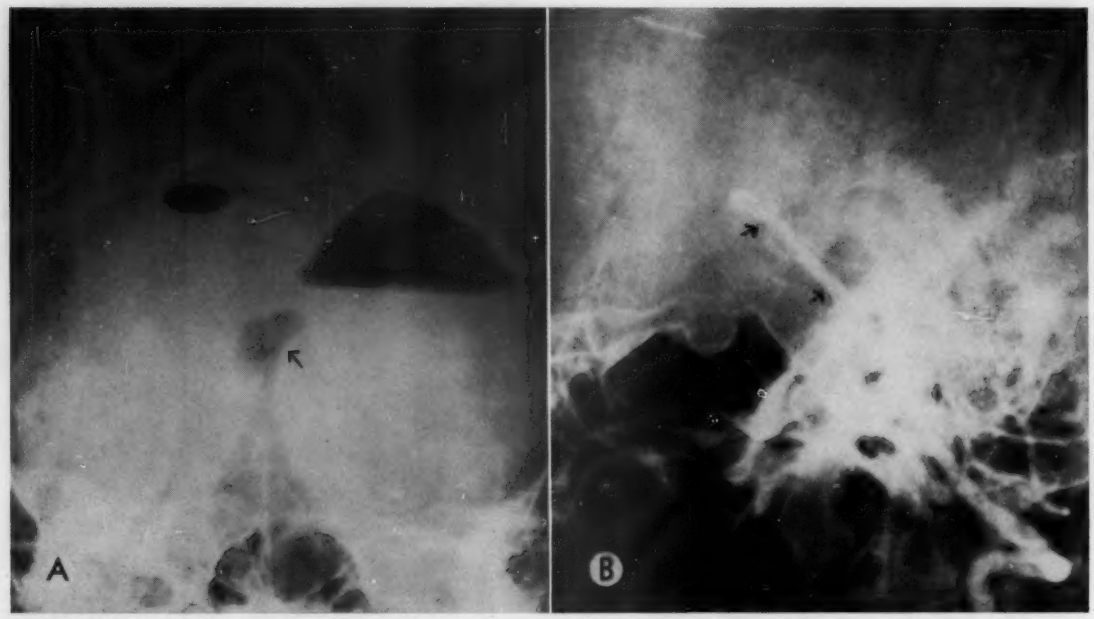

FIG. 11. *Anterolateral saddle tumor, meningioma, left gasserian-petrosal and tentorial attachment.* The patient, a sixty-six year old man (unit no. 137-76-53), complained of hearing loss in the left ear of two years' duration, progressive left hemiparesis and some degree of mental deterioration. The pneumoencephalogram (*A*) shows the presence of a sharp indentation on the posteroinferior portion of the third ventricle on the left side (arrow). The upper portion of the aqueduct is slightly off center to the right and the fourth ventricle returns almost to the midline in its caudal portion. A vertebral angiogram (*B*) shows moderate posterior displacement of the basilar artery particularly involving its upper portion and the bifurcation (arrows). The initial portion of both posterior cerebral and superior cerebellar arteries is displaced posteriorly, more on one side than on the other. The findings are typical of a tumor interposed between the clivus and the basilar artery.

There were no findings on plain roentgenograms in this case. High papilledema was present clinically. Ventriculography revealed sharp lateral displacement of the aqueduct and the usual indentation on the posteroinferior portion of the third ventricle (Fig. 15). There was also elevation of the floor of the lateral ventricle and lateral displacement of the temporal horn which presented a sharp rounded configuration on its medial portion similar to that seen in thalamic tumors. For this reason a thalamic mass was mistakenly suspected preoperatively.

IV. POSTEROLATERAL MASSES

These are situated, as the name indicates, posterolateral to the brain stem. They may arise from the tentorium or from the posterior cerebral artery. Only one of our cases belongs in this group, a large aneurysm thought to arise from the posterior cerebral artery; the aneurysm was found to be completely thrombosed at operation.

Pneumoencephalography revealed a deformity of the cisterna ambiens; the lateral wing of the cistern was displaced forward and had become concave backwards. There was also a mass compressing the medial portion of the atrium of the ventricle (Fig. 16, *A* and *B*). The posterior third ventricle was not indented. Vertebral angiography performed postoperatively showed no filling of the aneurysm which had been occluded surgically by imbricating its walls.

V. POSTERIOR INCISURAL MASSES

This group included 3 cases, all meningiomas, arising from the tentorial edge (one case has not been surgically verified). Pineal tumors, as mentioned above, may sometimes grow chiefly outside of the third ventricle but they are not included in this discussion. Aneurysmal saccular dilatation of the vein of Galen observed in 3 addi-

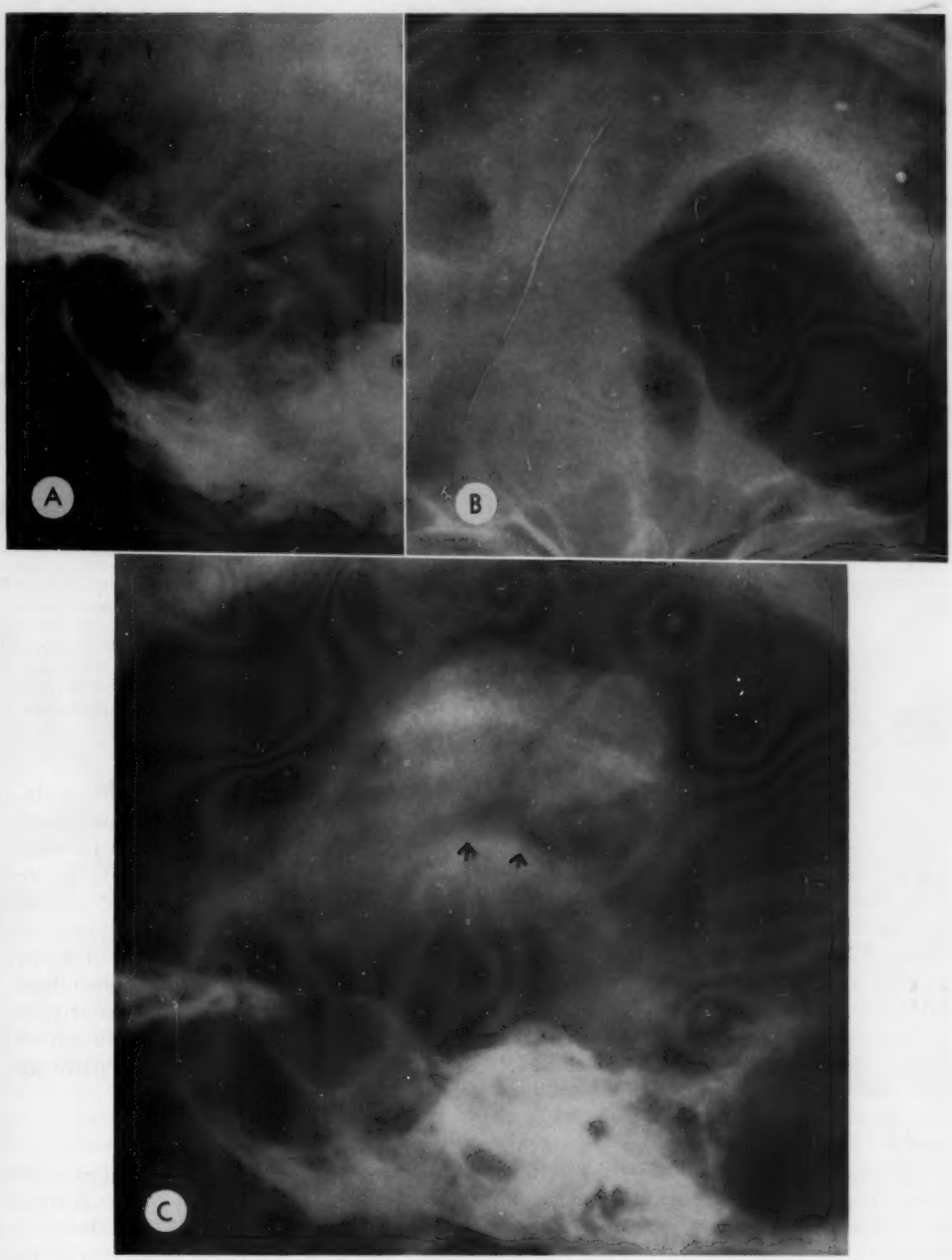

Fig. 12. *Anterolateral dumbbell (saddle) meningioma.* The patient, a forty-four year old woman (unit no. 20-10-58), complained of slightly decreased hearing and tinnitus in the right ear for one year, and progressive ataxia for two months. The plain roentgenogram (*A*) shows a diffuse sclerosis of the dorsum sellae and of the upper portion of the clivus. A ventriculogram in the frontal projection (*B*) reveals moderate ventricular dilatation. There is an indentation on the posteroinferior aspect of the third ventricle (arrow). The lateral ventriculogram (*C*) shows an elevation of the temporal horn (two arrows).

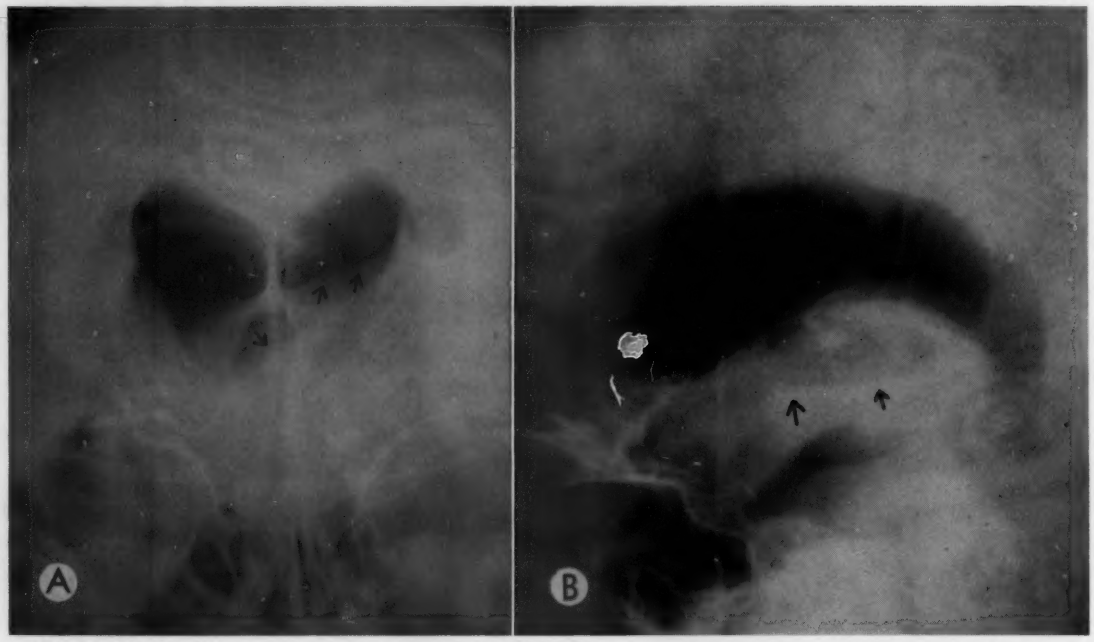

FIG. 13. *Anterolateral saddle meningioma showing elevation of the third ventricle.* The patient, a fifty-four year old woman (unit no. 139-16-58), complained of headaches, unsteady gait, tinnitus and decreased hearing on the left side, numbness of the left side of face and slight left hemiparesis. The plain roentgenograms showed a few specks of calcium in the region of the incisura on the left side. Ventriculography was performed because of evidence of increased intracranial pressure. The frontal ventriculogram (*A*) shows the presence of an indentation on the inferolateral aspect of the third ventricle on the left side (one arrow). There is elevation of the floor of the left lateral ventricle (two arrows). In the supine lateral ventriculogram (*B*) it is noted that the entire third ventricle is elevated also including its hypothalamic portion (arrows). Operation disclosed a meningioma attached to the medial portion of the floor of the middle fossa and extending into the posterior fossa through the incisura. Because the tumor was more medial than in the case shown in Figure 12, there is no elevation of the temporal horn which is normal in position. There is, however, elevation of the floor of the lateral ventricle.

tional cases belongs in this group. The latter have been reported elsewhere (Taveras and Poser[4]) and will not be discussed in detail.

The plain roentgenograms did not reveal any abnormalities other than increased intracranial pressure in one case.

Pneumoencephalography in the midline posterior tentorial meningiomas reveals obliteration or deformity of the cistern over the quadrigeminal plate associated with forward displacement and bowing of the quadrigeminal plate and of the aqueduct (Fig. 17, *A* and *B*). Because the mid-brain is flattened from behind there may be spreading of the lateral arms of the cisterna ambiens as seen in the frontal or reversed cerebellar projection (Fig. 17*B*). If the tumor is larger we may expect an indentation on the posterior aspect of the third ventricle similar to that produced by a pinealoma (Fig. 18, *A–D*). The aneurysmal dilatation of the vein of Galen produces an indentation on the posterior third ventricle similar to that of pinealoma. If the tumor projects above the tentorium, an indentation on the medial side of the atrium of the lateral ventricles may be observed such as is shown in Figure 18*B*.

In the meningiomas angiograms may be expected to demonstrate downward and forward displacement of the vein of Galen and the posterior portion of the internal cerebral vein (Fig. 19). If the tumor extends above the tentorial edge, it will produce spreading of the posterior cerebral arteries (Fig. 18, *C* and *D*). The aneurysmal dilatation of the vein of Galen is an

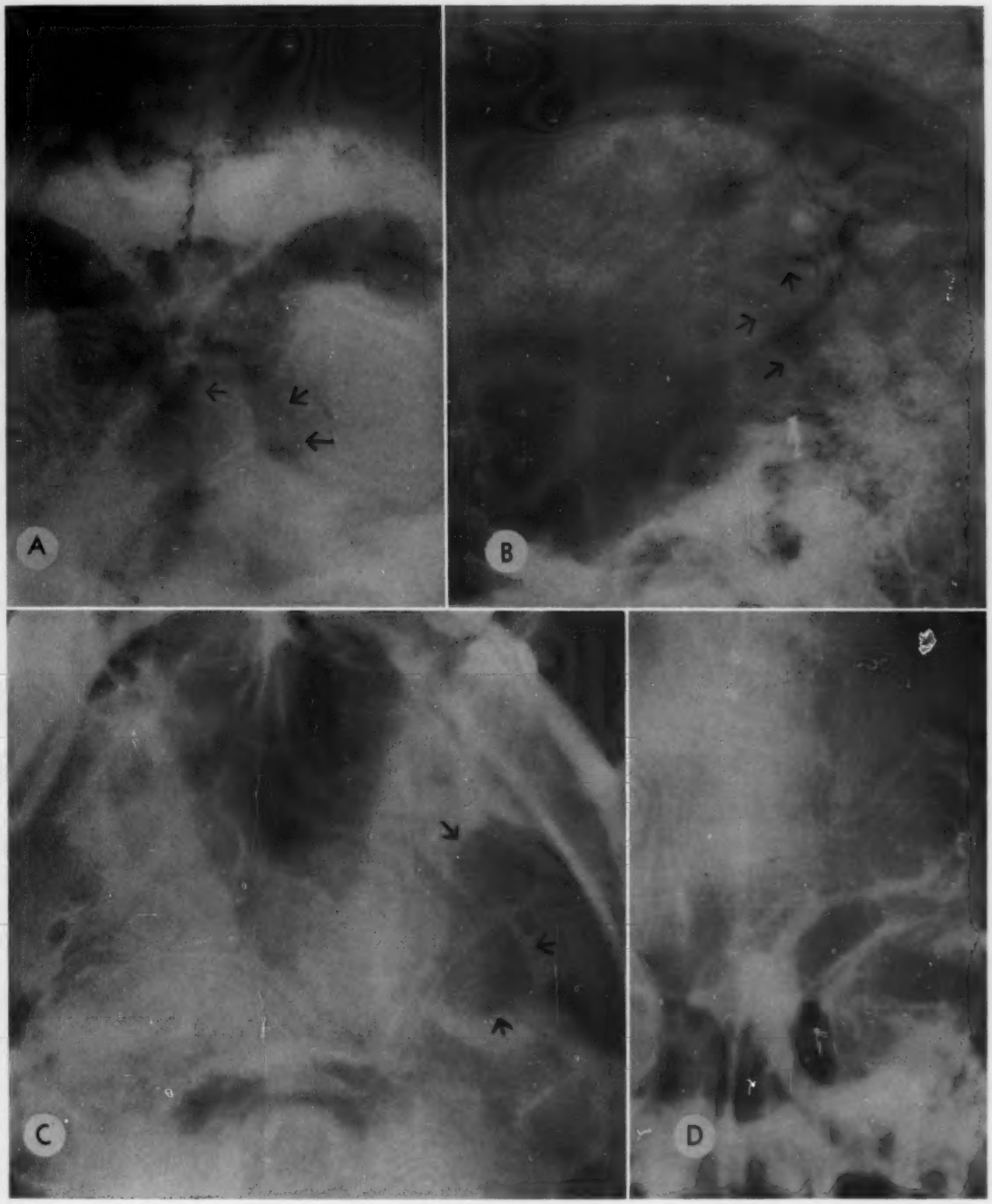

FIG. 14. *Anterolateral mass, saddle meningioma extending into middle fossa and posterior fossa anterolaterally.* The patient, a thirty-nine year old man (unit no. 139-94-41), gave a history of sudden double vision starting fifteen months before admission. The diplopia persisted with only partial clearing. Neurologic examination showed a third nerve palsy and decreased sensation in the territory of the first and second divisions of the fifth nerve on the left side. Pneumoencephalography was performed. The frontal projection (*A*) shows lateral deviation of the aqueduct (arrow). The fourth ventricle is almost in the midline. The left lateral arm of the cisterna ambiens is dilated (two arrows) in comparison to the opposite side, and presents a sharp cut-off at its lower portion. The lateral projection (*B*) shows an elevation of the lateral wing (retrothalamic portion) of the cisterna ambiens on the side of the tumor (upper arrow). The opposite side is in normal position. In addition, there is a collection of gas which evidently represents a dilated lateral arm of the cisterna ambiens over the tumor (two arrows). This is the same dilated space which is seen in the frontal reversed cerebellar projection on the left side (two arrows). The base view of the skull (*C*) shows evidence of bone destruction involving the floor of the middle fossa on the left side (two arrows). There is enlargement of the foramen ovale (arrow). Pneumoencephalography confirmed the presence of a tumor mass in the posterior fossa as well as in the middle fossa. A left carotid angiogram (*D*) shows moderate elevation of the horizontal portion of the middle cerebral artery and slight medial displacement of the supraclinoid portion of the internal carotid artery.

FIG. 15. *Laterally placed incisural mass (meningioma) simulating a thalamic tumor.* An eighteen year old girl (unit no. 132-17-26) was admitted to the hospital complaining of headaches of nine months' duration and weakness and tingling of her hands of five days' duration. Neurologic examination disclosed generalized weakness of the arms in all the muscle groups including the shoulders. There was evidence of cerebellar dysfunction. The fundi showed bilateral papilledema with multiple hemorrhages. The ventriculogram demonstrates the presence of a sharp indentation on the inferolateral aspect of the third ventricle in its posterior portion (upper arrow). There is a sharp displacement of the aqueduct to the right of the midline (two arrows); the fourth ventricle returns to normal position lower down. Other ventriculograms showed that the floor of the right lateral ventricle was elevated. Roentgenologically, a thalamic tumor was suspected and the possibility of a meningioma was only mentioned but not seriously considered. Surgical intervention disclosed a meningioma attached to the edge of the tentorium, which was completely removed

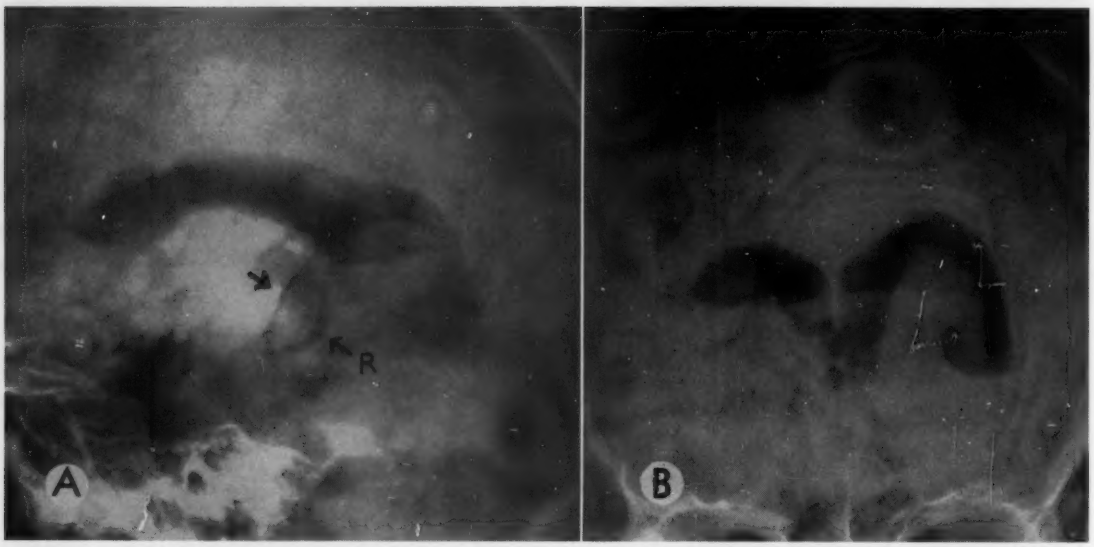

FIG. 16. *Posterolateral incisural mass, aneurysm arising from the posterior cerebral artery.* The patient was a forty-four year old white man (unit no. 11-87-71) who gave a history of waking up with a sensation of malaise and right hemianopsia one morning about two weeks prior to admission. The next day he was disoriented and amnesic. Blood pressure then was 200 systolic. A pneumoencephalogram in the lateral projection (*A*) reveals the presence of a mass lesion encroaching on the atrial portion of the lateral ventricles on the medial side. There is an asymmetry of the wings (retrothalamic portion) of the cisterna ambiens. The one on the right side is normal in position (labeled R with arrow on the figure), and the one on the left side is bowed upwards (upper arrow). The frontal pneumoencephalogram (*B*) discloses the sharply outlined mass encroaching on the atrial portion of the ventricles. The outline is so sharp that it was originally thought that this was a partly intraventricular tumor. Surgical intervention disclosed a thrombosed aneurysm. The mass was opened and emptied of clotted blood and the walls were plicated. Subsequently, vertebral angiography was carried out which disclosed distortion of the left posterior cerebral artery; a major branch of it was not outlined. No aneurysmal sac was filled.

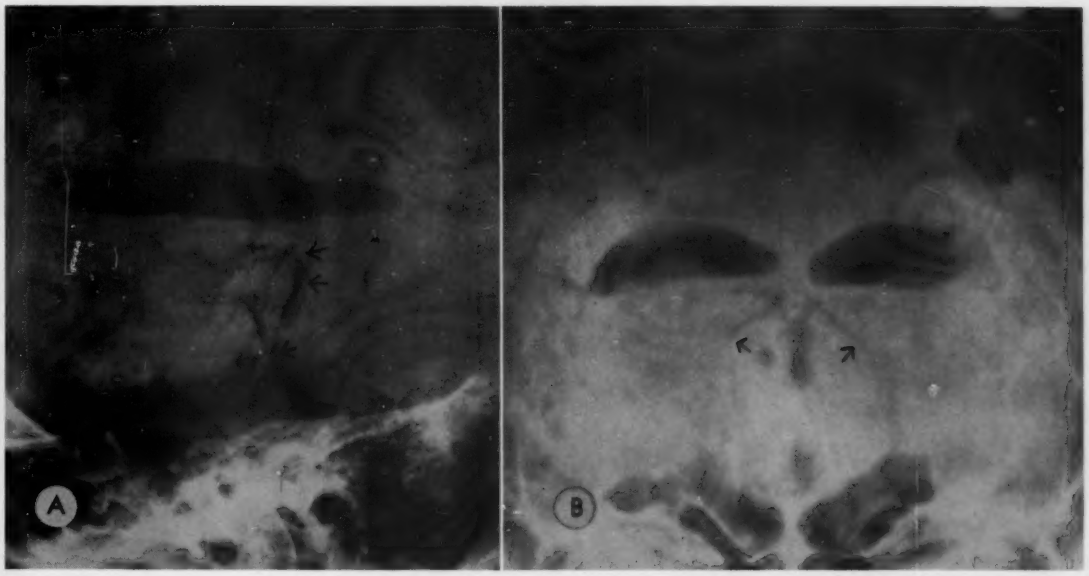

Fig. 17. *Posterior midline mass, malignant meningioma arising from the edge of the tentorium.* The patient, a nineteen year old man (unit no. 12-86-52), came into the hospital complaining of headaches of three to four months' duration, staggering gait, and speech difficulty of about four weeks' duration. Nausea, vomiting and subjective vertigo appeared the week before admission. The pneumoencephalogram in the lateral projection (*A*) demonstrates a forward angulation of the aqueduct (lower arrow) and a distortion of the lower portion of the quadrigeminal cistern. The quadrigeminal plate is distorted and convex upwards to a marked degree (upper two arrows). In the frontal projection (*B*) there is widening of the air space around the quadrigeminal plate with a disappearance of the normal appearance of the quadrigeminal tubercles (arrows).

arteriovenous communication in which there is marked saccular enlargement of this vein. Angiography is diagnostic.

DISCUSSION

It is apparent from the foregoing that it is usually possible to arrive at a correct diagnosis of an extra-axial incisural mass by roentgen methods. In the majority of cases pneumoencephalography will be the procedure of choice. Cisternal as well as ventricular fillings are easily obtained in these cases and carry no danger to the patient if performed carefully. In our series of cases ventriculography was performed only in the presence of severe increased intracranial pressure (3 cases) or when no ventricular filling was obtainable by pneumoencephalography (2 cases).

Vertebral angiography and sometimes also carotid angiography are required whenever the possibility of a vascular lesion is raised. Angiography is particularly important in the midline anterior lesions, not only because many of them are basilar artery aneurysms, but also because the posterior displacement of the basilar artery is diagnostic of an extra-axial anterior mass (Fig. 11*B*).

Although aneurysm of the posterior cerebral artery can never be excluded with certainty without angiography, we have not been performing angiography routinely in the anterolateral masses, the largest group. Bulky aneurysms arising from this portion of the posterior cerebral artery are very rare. Angiography is performed if the history presents a sudden onset, or in cases where additional information is required before surgical intervention.

The differential diagnosis is as follows:

1. An anterior midline mass may be confused with an intra-axial brain stem tumor. In the latter instance the diagnosis of an extra-axial mass may be made if there is air in the cisterns around the tumor which would reveal that it is separate from the brain stem (Fig. 5; and 6*B*). If no air is present here, vertebral angiography will demonstrate posterior displacement of the

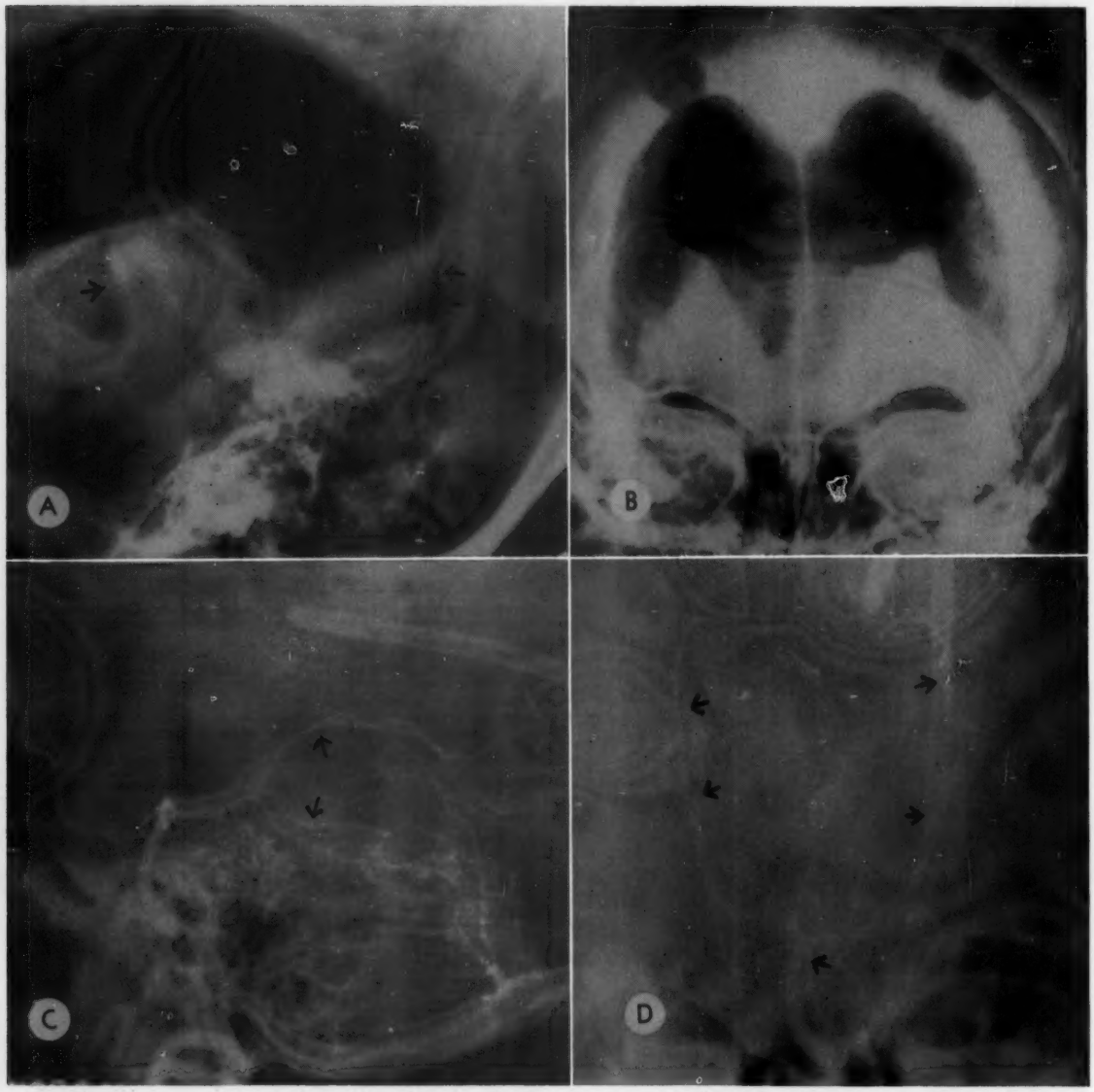

Fig. 18. *Midline posterior mass, large meningioma arising from the tentorial edge.* A forty-seven year old woman (unit no. 138-03-44) complained of blurring of vision for over a year, occipital headaches for eight months, and slight ataxia. There was bilateral papilledema and a partial right homonymous hemianopsia. Patient had marked increased intracranial pressure. The plain roentgenograms showed destruction of the sella turcica. Ventriculography was performed which revealed a block at the posterior third ventricle with a mass obstructing this portion of the third ventricle. The appearance suggested pinealoma. The patient was then placed in the sitting position and a small amount of gas was injected via the lumbar route. The lateral pneumoencephalogram (*A*) reveals a large mass encroaching on the posterior portion of the third ventricle (single arrow) and also encroaching on the subarachnoid space over the surface of the cerebellum (double arrow). The diagnosis of pineal teratoma was seriously entertained at this time. In the postero-anterior prone projection (*B*) there is evidence of an indentation on the inner aspect of the atrium of the lateral ventricle on each side (arrows). This indicates a supratentorial extension of the mass. A pineal teratoma could cause this but it is more likely for a tentorial meningioma to produce this type of deformity. Biopsy disclosed a meningioma; it was considered inoperable. A Torkildsen shunting procedure was then carried out and, later, vertebral angiography was performed. The lateral angiogram (*C*) shows the presence of a symmetric distortion of the branches of the posterior cerebral arteries. There is elevation of the internal occipital (parieto-occipital) branch of the posterior cerebral on each side (upper arrow) and slight depression of the temporo-occipital (posterior temporal) branches (lower arrow). The frontal angiogram (*D*) shows marked separation of the branches of the posterior cerebral artery which normally reach the midline after they turn around the mid-brain (arrows). The bifurcation of the basilar artery is indicated by the lower arrow. An attempt at a partial removal of this tumor resulted in the patient's demise. She had continued to run a down-hill course following the initial shunting procedure.

Fig. 19. *Posterior midline mass, probably meningioma* (not proved as yet). A seventy-eight year old woman (unit no. 22-99-14) had been complaining of gait disturbance for four years and loss of memory for recent events. She had left homonymous hemianopsia and weakness of the left upper extremity. Pneumoencephalography failed to produce any ventricular filling. There was evidence of distortion of the cistern of the quadrigeminal plate. Carotid angiography was performed which revealed no abnormal findings in the arterial phase. The angiogram in the venous phase shows a downward displacement of the vein of Galen and a forward and downward displacement of the posterior portion of the internal cerebral vein (arrows). It would be difficult to explain this type of displacement in addition to the distortion noted in the air study by a lesion other than a meningioma arising from the edge of the tentorium. The history was very slowly progressive and did not indicate a corpus callosum tumor involving particularly the splenium which could produce similar angiographic findings.

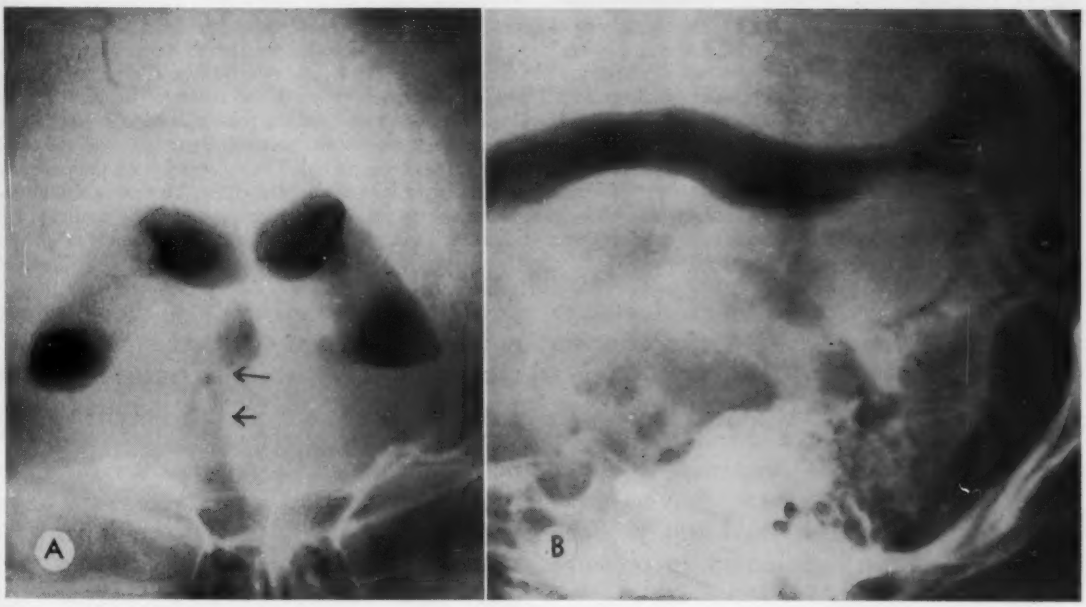

Fig. 20. *Brain stem tumor simulating an extra-axial mass.* The patient, a fourteen year old boy (unit no. 142-11-60), had a history which was compatible with a brain stem tumor although slightly atypical. Because of the presence of papilledema and signs of increased intracranial pressure, ventriculography was performed. The aqueduct and fourth ventricle were not well visualized. The pneumoencephalogram in the frontal projection (*A*) shows displacement of the aqueduct and fourth ventricle to the right of the midline (arrows). However, the fourth ventricle gives more the impression of being encroached upon rather than simply displaced and rotated as is the case in extra-axial lesions. The lateral projection (*B*) shows a typical rounding and posterior displacement and "stretching" of the aqueduct and fourth ventricle which were considered to be due to brain stem tumor although an extra-axial ventrally placed lesion could produce similar findings. No surgical intervention was deemed necessary and radiation therapy resulted in marked improvement of the neurologic findings within four weeks.

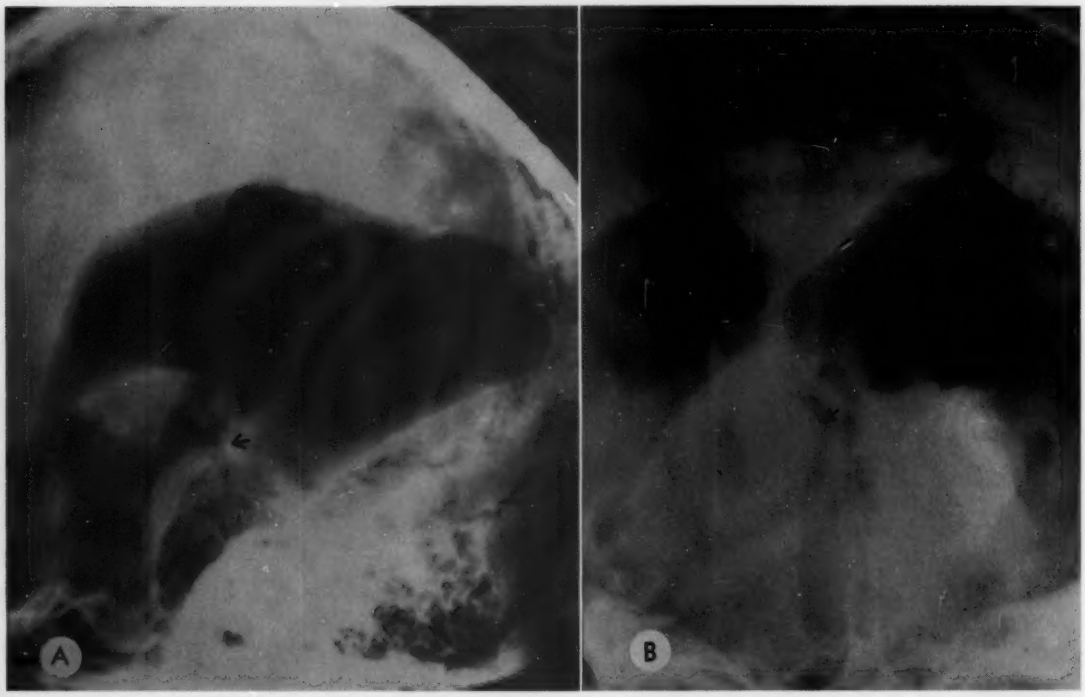

Fig. 21. *Large right cerebellar tumor with upward herniation and elevation of the right tentorial region.* The patient, a fifteen year old girl (unit no. 29-37-95), had been complaining of difficulty in walking for several months. Neurologic examination revealed typical cerebellar ataxia, diminished vision in the left eye with obvious bilateral papilledema with exudates. Ventriculograms demonstrated a moderate degree of ventricular dilatation. The lateral view (*A*) shows a marked degree of forward displacement of the aqueduct (single arrow). There is moderate elevation of the atrium of the right lateral ventricle as well as of the lower aspect of the occipital horn in comparison to that of the other side (double arrow). In the posteroanterior prone projection (*B*) there is evidence of an encroachment on the posteroinferior aspect of the third ventricle (arrow). At operation a large right cerebellar astrocytoma was encountered. The indentation on the posterior third ventricle was evidently produced by upward herniation of the right lobe of the cerebellum. There was also elevation of the atrium which was produced by marked bulging of the tentorium. The combination of these findings was very suggestive of a tentorial meningioma growing downwards as well as upwards. The age of the patient was against the latter diagnosis, however.

basilar artery in the extra-axial masses (Fig. 11*B*) and anterior displacement or no displacement of the basilar artery in brain stem tumors. It should be remembered that the presence of gas in the cisterna pontis and interpeduncularis does not rule out tumor here because the tumor may be laterally placed or there may be atrophy of the adjacent structures secondary to pressure by the tumor.

2. The anterolateral single and the direct lateral tumors can be confused with intra-axial tumors involving the mid-brain and the thalamus. Usually brain stem tumors are not situated strictly to one side of the aqueduct but are mostly rostral to the aqueduct of Sylvius. Therefore, they produce posterior displacement of the aqueduct and only occasionally do they produce any significant lateral aqueductal displacement. One such case is illustrated in Figure 20, *A* and *B*. A tumor centered primarily in the thalamus and extending down into the mid-brain may also produce similar findings. In this case, however, there is apt to be flattening or indentation on the upper portion of the third ventricle, whereas the extra-axial incisural masses will indent only the posteroinferior portion.

The anterolateral dumbbell tumors can usually be diagnosed with certainty if one observes the changes involving the temporal horn as well as the typical deformity of the inferolateral aspect of the third ventricle, which in these cases may also involve the anterior portion of this ventricle

(Fig. 12, *B* and *C*). Findings on plain roentgenograms are more common in this group.

3. The posterior midline and posterolateral masses may be difficult to differentiate on air studies from high cerebellar tumors growing towards the midline. Large cerebellar tumors accompanied by upward herniation through the incisura may mimic a posterolateral mass. There may even be elevation of the atrium of the lateral ventricle suggesting a tentorial origin (Fig. 21, *A* and *B*). It may be difficult or impossible in some cases to differentiate between a midline posterior extracerebral mass (either meningioma or aneurysmal dilatation of the vein of Galen) and a pinealoma by air studies. Angiography will be of help here; in meningioma from the tentorial edge the vein of Galen and the posterior portion of the internal cerebral vein will most probably be displaced downward and forward (Fig. 19). In pinealoma the internal cerebral vein is apt to be either normal or somewhat displaced upward in its posterior portion. Vertebral angiography may add further information, particularly if there is upward extension of the tumor (Fig. 18, *C* and *D*).

SUMMARY

A group of 24 patients with masses in the region of the incisura of the tentorium has been presented. The cases were subdivided into five groups (midline anterior, anterolateral, lateral, posterolateral and midline posterior) depending upon their relationship with the mid-brain. Only the extra-axial masses were thought properly to belong in this discussion. Sixteen of the cases were meningiomas, 5 were aneurysms, and 3 were chordomas. Three additional cases of aneurysmal dilatation of the vein of Galen were mentioned but not discussed. The diagnosis is usually made by pneumoencephalography, but vertebral and sometimes carotid angiography may also be required. In most of the cases, except those of the midline group, the posteroinferior portion of the third ventricle presented a fairly characteristic indentation as seen in the frontal posteroanterior projection, and the aqueduct was displaced laterally. Surgical intervention must be based on a very accurate anatomic diagnosis. In the dumbbell or saddle type of tumor a combined approach through the posterior and middle fossae may be required.

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REFERENCES

1. Castellano, F., and Ruggiero, G. Meningiomas of posterior fossa. *Acta radiol.*, 1953, Suppl. 104, p. 119.
2. Cushing, H., and Eisenhardt, L. Meningiomas: Their Classification, Regional Behaviour, Life History, and Surgical End Results. Charles C Thomas, Publisher, Springfield, Ill., 1938.
3. Jefferson, G. Trigeminal neurinomas with some remarks on malignant invasion of gasserian ganglion. *Clin. Neurosurg.*, 1955, *1*, 11–54.
4. Taveras, J. M., and Poser, C. Roentgenologic aspects of cerebral angiography in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, 82, 371–391.

INTRACRANIAL SARCOMAS: THEIR ROENTGENOGRAPHIC MANIFESTATIONS*

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SARCOMAS that arise primarily within the cranial cavity are uncommon neoplasms, the differential classification of which has engendered controversy among neuropathologists ever since they were defined by Bailey,[2] in 1929. In the main, this controversy has concerned the origin of the cells that constitute primary sarcomas of the brain. Some authors maintain that the embryonic origin of the meninges and of the pial and cerebral vessels and their adventitia is ectodermal, whereas others have concluded that they are of mesodermal origin. If the former contention were correct, these tumors would not be sarcomas.

Because no embryologist today espouses the outmoded theory of the specificity of the germ layers, Abbott and Kernohan[1] concluded that neither of the aforementioned opinions was of much value and that the best approach to evaluation of these tumors was the direct study of their histologic structure. Such a study reveals that all of these neoplasms are of the connective-tissue type (sarcomas), regardless of the germ layer from which they supposedly arose. A similar opinion was expressed by Hsü.[9]

At the present time, Kernohan and Uihlein[12] are reviewing all cases of primary intracranial sarcomas recorded in the files of the Mayo Clinic. They have concluded that all these neoplasms may be classified into one of five categories; namely, fibrosarcoma, perivascular sarcoma, reticulum cell sarcoma, hemangiopericytoma, and a miscellaneous group. It is beyond the scope of this paper to discuss the histopathologic details of these neoplasms; suffice it to say that the tumors in each category have a distinctive microscopic appearance that enables the trained neuropathologist to differentiate them.

REVIEW OF THE LITERATURE

Intracranial sarcomas have been almost universally ignored in the radiologic literature. I was unable to find a single article dealing with the general roentgenologic aspects of these neoplasms. Some of the textbooks of roentgenologic and neuroroentgenologic diagnosis[5,19] mention that such lesions occur, but little pertinent information beyond this is provided.

Most writers on this subject, who are generally neurosurgeons and neuropathologists, mention roentgenologic findings only briefly as portions of case reports.[3,4,7,8,10,11,13–18,20–27] From these brief references, it may be presumed that in most cases the plain roentgenogram of the skull was normal, although occasionally pineal shift or sellar decalcification was observed. On rare occasions, the tumor was partially calcified. Cerebral pneumography or angiography or both enabled the expanding mass to be accurately localized in almost all instances.

Lorenz,[15] as well as Milletti,[17] stated that the arteriographic appearance of these neoplasms is characteristic. Many large undulating vessels are seen in the center of an ill-defined zone occupied by tumor. Arteriovenous fistulas, vascular lakes and a few capillary networks also are seen frequently. If these authors could demonstrate that a portion of this blood supply was derived from the external carotid artery, they considered that the unequivocal diagnosis of sarcoma could be made. If no such connection could be demonstrated, they considered the tumor to be a glioblastoma multiforme, as the vascular pattern was the

* Presented at the Sixtieth Annual Meeting of the American Roentgen Ray Society, Cincinnati, Ohio, September 22–25, 1959.

same as that observed in the latter neoplasm.

MATERIAL AND CLINICAL DATA

As no roentgenographic material on this subject was available prior to 1945, all cases analyzed herein were accumulated subsequent to that year. A search of the records that concern tumors of the brain at the Mayo Clinic yielded 54 cases of neoplasms that could be considered to come within the confines of this survey. These 54 tumors included 21 perivascular sarcomas, 19 fibrosarcomas, 6 reticulum cell sarcomas, 5 hemangiopericytomas, and 3 miscellaneous sarcomas (chondrosarcoma, myxochondrosarcoma and osteochondrofibromyxosarcoma). The patients ranged in age from two months to sixty-five years. With the exception of the second decade of life, in which there were only 3 patients, the distribution according to decades was about equal; 10 patients were in the first decade, 9 each in the third, fourth and sixth decades, and 7 each in the fifth and seventh. The average age was thirty-six years. As almost 80 per cent of the neoplasms occurred in patients more than twenty years of age, it may be said that these are tumors of adults.

The series included 27 male patients and 27 female. The majority of the perivascular sarcomas were found in females and the majority of fibrosarcomas in males (Table 1).

A total of 45 tumors arose in the supratentorial portion of the skull, whereas the remaining 9 were located infratentorially. Their rate of growth varied clinically from slow to extremely rapid. Therefore, the presenting symptoms and neurologic signs also varied depending on the rapidity of growth and the situation of the mass.

Table I

SEX DISTRIBUTION OF 54 PRIMARY INTRACRANIAL SARCOMAS

Type of Tumor	Sex of Patient	
	Male	Female
Perivascular sarcoma	4	17
Fibrosarcoma	16	3
Reticulum cell sarcoma	3	3
Hemangiopericytoma	3	2
Miscellaneous	1	2
Total	27	27

ROENTGENOLOGIC FINDINGS

Plain Roentgenograms. Routine roentgenograms of the skull, available in 53 cases, revealed a lack of positive findings in only 16. In the remaining 37 (70 per cent), signs of an intracranial neoplasm were discernible both directly, as evidenced by the presence of calcium within the lesion or the invasion and destruction of adjacent bony structures by the mass, and indirectly, as evidenced by displacement of a calcified pineal gland or choroid plexus, separation of cranial sutures, or decalcification about the sella turcica secondary to generalized increased intracranial pressure.

The presence of abnormal roentgenologic findings was most conspicuous among the fibrosarcomas (15 of 19 cases), reticulum cell sarcomas (5 of 6 cases), and the miscellaneous tumors (all 3 cases). The perivascular sarcomas (11 of 20 cases) and hemangiopericytomas (3 of 5 cases) were somewhat more disappointing in this regard.

The pineal gland was calcified and visible in 6 of the 16 "negative" roentgenograms and in 23 of the 37 "positive" roentgenograms. In this latter group, it was deviated in 14 cases, and lateral displacement was observed in all 14; in addition, the glands in 4 cases were also displaced in the anteroposterior or the superoinferior direction. The choroid plexus was displaced superiorly and medially in 2 additional cases in which the pineal gland was not visible.

The presence of increased intracranial pressure was manifested by separation of the cranial sutures in 11 cases and by decalcification of the sella turcica, sometimes accompanied by separation of the sutures, in 14 cases.

The position of the neoplasm was directly

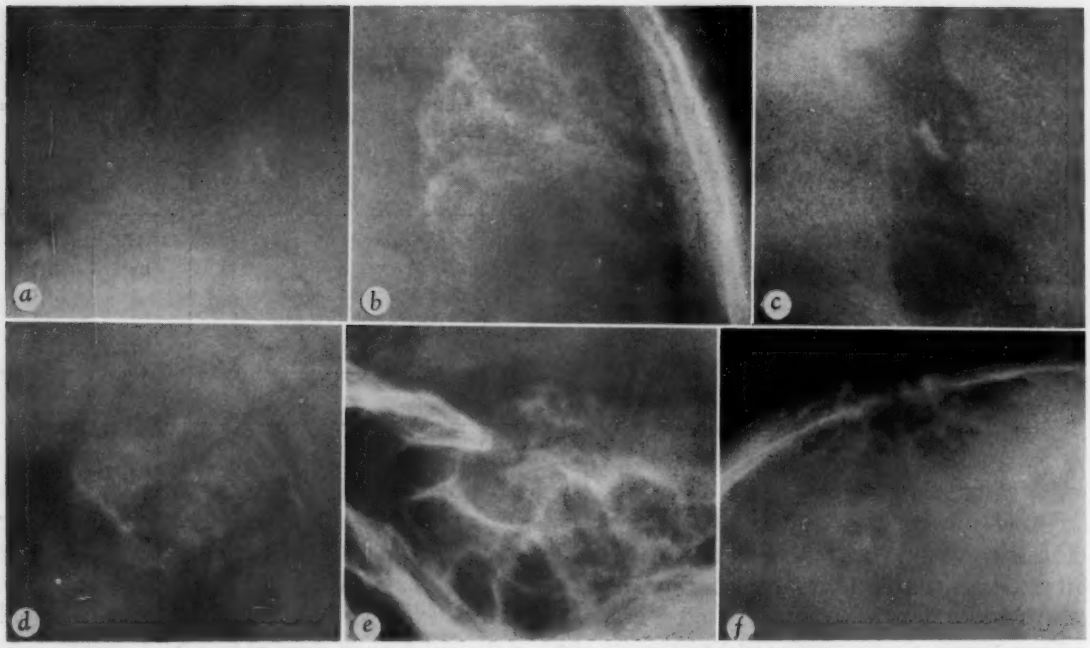

Fig. 1. Calcification within the substance of 6 different intracranial sarcomas. Note similarity in appearance among many of them. (*a*, *b* and *c*) Fibrosarcomas. (*d*) Osteochondrofibromyxosarcoma. (*e*) Chondrosarcoma. (*f*) Fibrosarcoma with invasion of bone in addition to calcification.

indicated in 11 cases. Zones of calcification within the mass were observed in 5 instances, and invasion and destruction of adjacent bony structures were seen in a similar number. Both of these characteristics appeared in the remaining case. The zone of calcification was amorphous and conglomerate in nature, measured 1 to 4 cm. in diameter, and was remarkably similar in appearance in all cases, although not specifically diagnostic in nature (Fig. 1). Four of the calcium-containing tumors were fibrosarcomas, one a chondrosarcoma, and one an osteochondrofibromyxosarcoma. The bony lesions were all lytic in nature and were produced by 3 fibrosarcomas and 3 perivascular sarcomas. The predominant appearance was that of diffuse lysis of bone which was rather widespread. In one case, the malignant cells apparently invaded the diploë, causing expansion and somewhat spotty destruction (Fig. 2).

A summary of these findings is found in Table II.

Pneumography. Air contrast studies,

Table II

Summary of findings* in plain roentgenograms of the head in intracranial sarcoma (53 cases)

Findings	No. of Cases
Pineal displacement (23 glands visible)	
None	9
Lateral only	10
Lateral and anteroposterior or superoinferior	4
Displacement of choroid plexus	2
Sellar changes	
None	19
Decalcification due to increased intracranial pressure	14
Calcified parasellar tumor	1
Separation of sutures	11
Tumor calcification	6
Bony invasion and destruction	6

* Positive findings present in 37 cases (70 per cent).

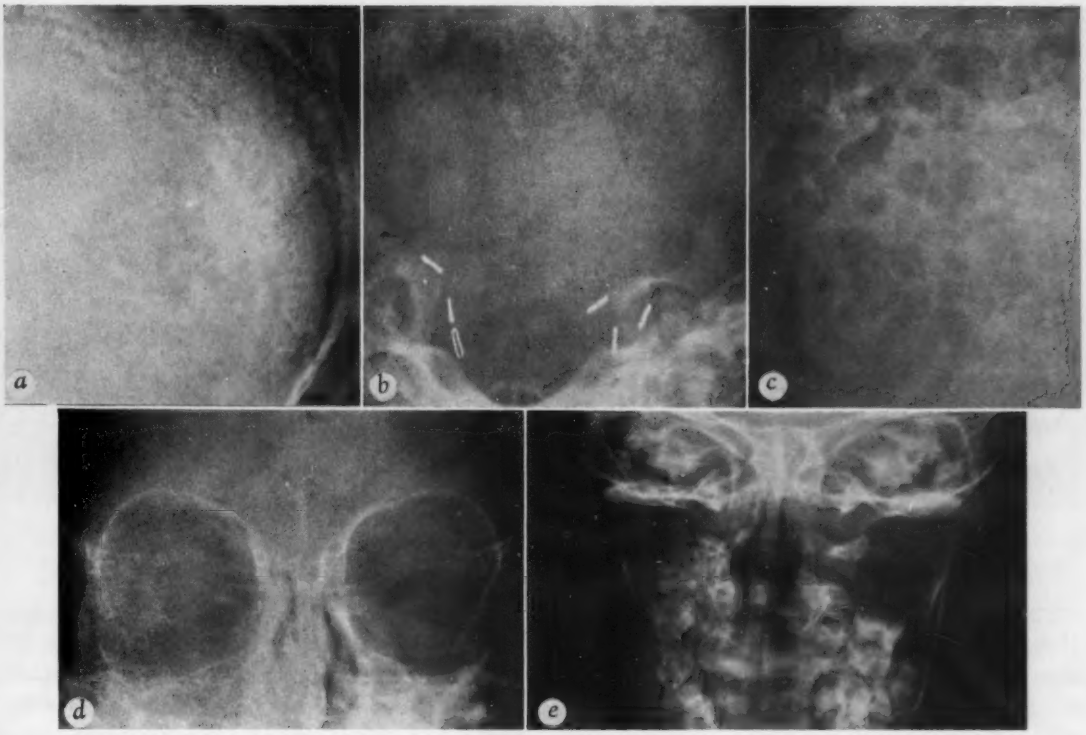

Fig. 2. Invasion and destruction of adjacent bony structures by intracranial sarcomas. (*a*) Perivascular sarcoma with tiny lytic zones in right frontal bone. (*b*) Fibrosarcoma invading and destroying right edge of old suboccipital operative defect. (*c*) Fibrosarcoma with mottled zones of destruction in calvarium (same case as Figure 1*f*). (*d* and *e*) Perivascular sarcoma invading left orbit and base of skull. Note downward displacement of upper molars.

either pneumoencephalography or ventriculography, were done in 27 of these patients, and it was possible to localize the lesion in the brain by the usual signs in all cases. As would be expected, precise localization of the supratentorial tumors was possible more frequently, but lateralization was possible in 4 of the 6 infratentorial lesions examined by these methods.

Air was introduced into two large cystic perivascular sarcomas by means of the ventriculographic needle. The walls of both cystic cavities were smooth except at the location of the tumor nodule. These nodules were 2 and 5 cm. in diameter, respectively, and had ragged irregular surfaces. Although this appearance was striking, it was not sufficiently unique to permit differentiation of cystic sarcomas from other cystic neoplasms of the brain.

A relatively frequent pneumographic finding and one that I believe is somewhat characteristic of sarcomas of the brain is the actual invasion of the ventricular walls by the neoplasm. This was observed in 9 of the 27 tumors considered in this section, including 6 perivascular sarcomas and one fibrosarcoma, one reticulum cell sarcoma and one hemangiopericytoma. At the site of invasion, the usual smooth surface of the ventricular wall was replaced by a rough, ragged-appearing zone. In some instances, the caliber of the involved ventricle was diminished (Fig. 3), whereas the appearance in other cases suggested that a cystic neoplasm had evacuated its contents into the ventricle (Fig. 4).

Carotid Angiography. The approximate position of the tumor could be ascertained in all but one of the 10 carotid angiograms that were made. The single exception was in the case of a small right parasellar fibro-

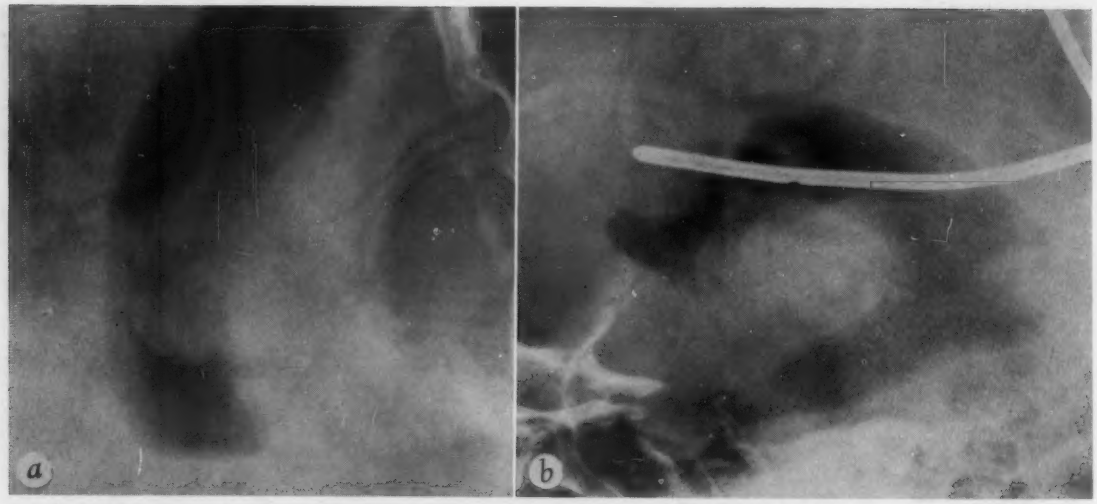

Fig. 3. (*a* and *b*) Neoplastic invasion of lateral ventricles by intracranial perivascular sarcomas. Note irregularity of the surface of the tumors. The view in *a* is a brow-up lateral view.

sarcoma in which the angiogram was made three months prior to the time that invasion and destruction of the adjacent bone by the neoplasm finally disclosed its position.

The precise localization of 4 of the remaining 9 tumors was made possible by the presence of abnormal vascularity in the neoplasm itself. The pathologic vessels had irregular outlines and edges that were not sharply demarcated. Vascular lakes, tangles of tiny vessels, and arteriovenous shunts were seen in all instances. The region of abnormal vascularity was never sharply demarcated from the normal surroundings (Fig. 5). These characteristics are the same as those usually associated with gliomatous tumors of the brain.

Definite participation of the external carotid arterial system in the blood supply

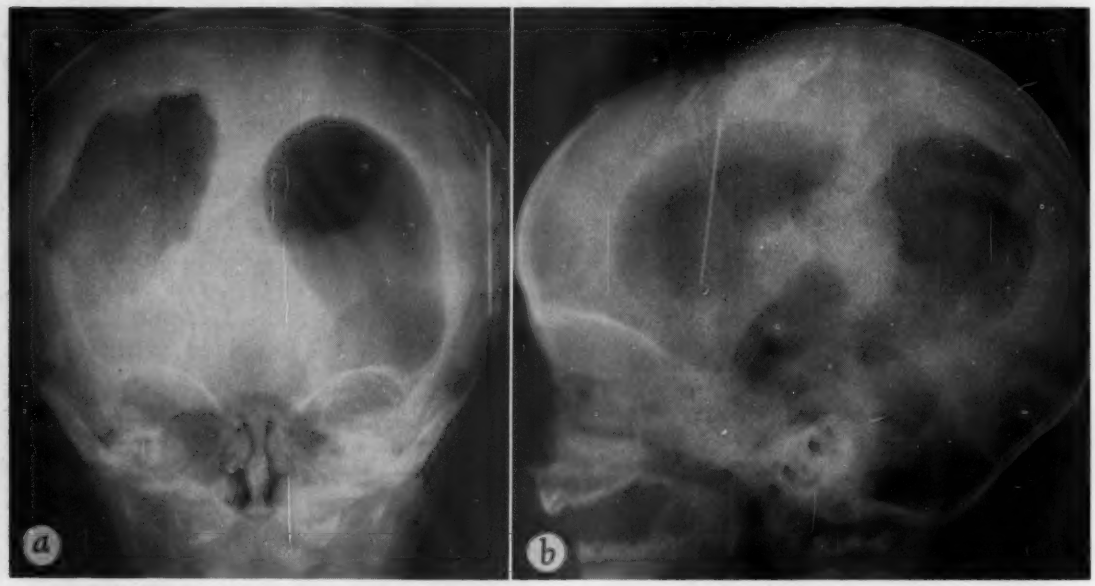

Fig. 4. (*a* and *b*) Two views of an intracranial reticulum cell sarcoma, showing spontaneous evacuation of a cystic tumor into the lateral ventricle. Note the irregular character of the cyst wall.

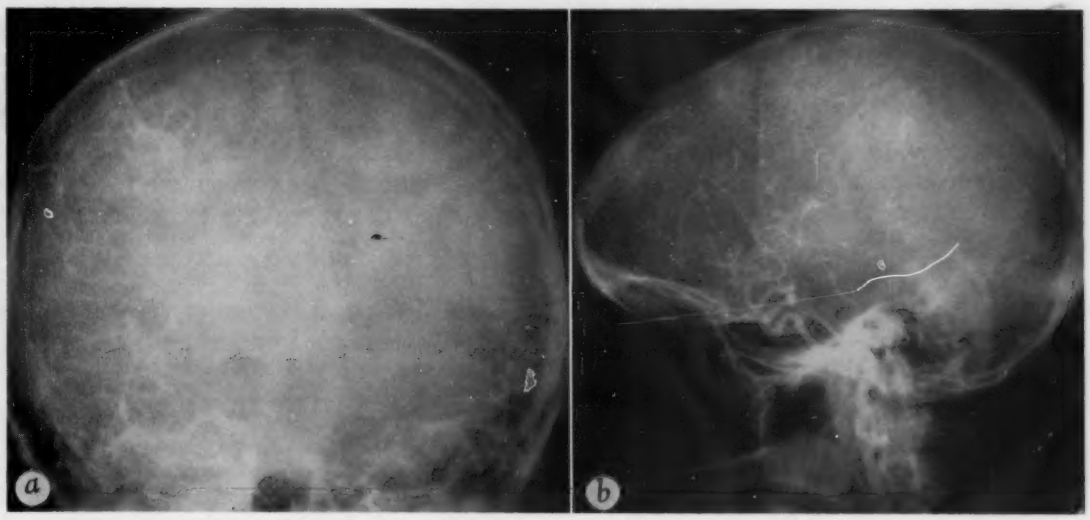

Fig. 5. (*a* and *b*) Carotid angiogram demonstrating abnormal tumor vascularity in a perivascular sarcoma in the right parietal region. Note irregular outline of vessels, vascular lakes and arteriovenous shunts.

of the tumor was demonstrated in 2 cases. These tumors were rather vascular and the arteries supplying them were moderately dilated. Figure 6 demonstrates one such neoplasm, with dilated arteries visible in the overlying scalp.

COMMENT

Roentgenologic evidence of intracranial sarcoma was lacking in only 4 of the 54 cases in this series. Most findings were nonspecific in nature but two signs appeared to be somewhat unique, and their presence would lend strong statistical support to a specific diagnosis of sarcoma.

One of these more specific signs is evidence of neoplastic invasion of the ventric ular wall, demonstrable by pneumography. I have seen only 2 of many hundreds of gliomas that demonstrated this particular finding and was able to find only 3 others mentioned in the literature.[5,6] However, one-third of the present group of sarcomas studied pneumographically disclosed evidence of such invasion. This finding then, although not absolutely diagnostic, would appear to be sufficiently suggestive to warrant the tentative diagnosis of sarcoma in those cases in which it is observed.

The other sign is seen in the carotid angiogram and consists of a "gliomatous type" of abnormal vascularity in the neoplasm contributed to by vessels of both the internal and external carotid arterial systems. As mentioned previously, this finding was described by Lorenz[15] and by Milletti,[17] and I have observed it only in the presence of an intracranial sarcoma. It is of less usefulness than is the aforementioned pneumographic sign in that the external carotid

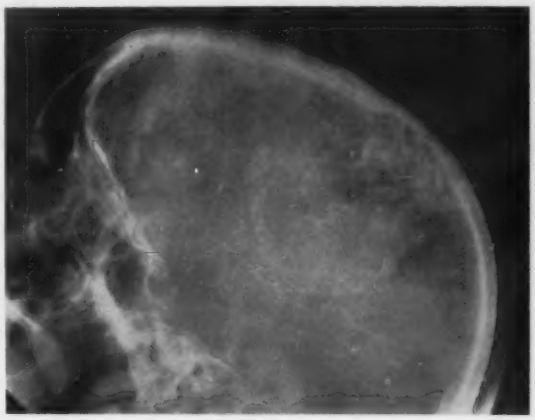

Fig. 6. Carotid angiogram demonstrating abnormal tumor vascularity in an intracranial hemangiopericytoma contributed to by branches of both the internal and external carotid arteries. Note dilated arteries in scalp over tumor zone.

arterial system often is not well visualized in the usual cerebral angiogram.

SUMMARY AND CONCLUSIONS

A group of 54 cases of primary intracranial sarcoma at the Mayo Clinic was studied to ascertain the character of the roentgenologic findings in such tumors. Some roentgenographic change was present in all but 4 of these cases.

A total of 70 per cent of the plain roentgenograms of the skull were abnormal. The observed abnormalities were nonspecific in character, consisting of shift of the pineal gland or choroid plexus, sellar decalcification, separation of sutures, calcification within the tumor, or invasion and destruction of bone.

Pneumographic studies, undertaken in 27 cases, revealed the location of the mass in all instances.

Carotid angiography failed to localize the neoplasm in only one of 10 cases. In 4 of the remaining 9 cases, exact localization was made possible by the appearance of abnormal vascularity within the tumor itself.

It is suggested that the observation of either of two findings may permit the radiologist to make the preoperative diagnosis of sarcoma with some assurance. The pneumographic demonstration of neoplastic invasion of the ventricular wall characterized by spontaneous communication of the ventricle with an adjacent cyst that has an irregular ragged lining or replacement of the usual smooth ventricular wall by a similar zone of irregularity is one of these findings. The other is the angiographic demonstration of both internal and external carotid arterial sources of blood to a tumor that exhibits pathologic vascularity of the type usually associated with gliomas of the brain.

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ADDENDUM

Since the completion of this manuscript, 4 additional cases of intracranial sarcoma have been observed. Two of these tumors were perivascular sarcomas and 2 were hemangiopericytomas.

Increased vascularity in a limited area of the skull overlying the tumor was observed in one case of hemangiopericytoma. This particular change had not been previously noted in the series.

The preoperative diagnosis of sarcoma, based on the observation of ventricular invasion, was made in another case.

REFERENCES

1. Abbott, K. H., and Kernohan, J. W. Primary sarcomas of the brain: Review of the literature and report of twelve cases. *Arch. Neurol. & Psychiat.*, 1943, *50*, 43–66.
2. Bailey, P. Intracranial sarcomatous tumors of leptomeningeal origin. *Arch. Surg.*, 1929, *18*, 1359–1402.
3. Christensen, E., and Lara, D. E. Intracranial sarcomas. *J. Neuropath. & Exper. Neurol.*, 1953, *12*, 41–56.
4. Cottrell, L. Primary fibrosarcoma of the brain. *Arch. Path.*, 1939, *27*, 895–901.
5. Davidoff, L. M., and Epstein, B. S. The Abnormal Pneumoencephalogram. Lea & Febiger, Philadelphia, 1950.
6. Earle, K. M., Snodgrass, S. R., Kuri, J., and Schneider, M. Cellular ependymoma with oligodendrial cells and multiloculation in the frontal lobe. *Cancer Sem.*, 1957, *2*, 58–60.
7. Globus, J. H., Levin, S., and Sheps, J. G. Primary sarcomatous meningioma (primary sarcoma of the brain). *J. Neuropath. & Exper. Neurol.*, 1944, *3*, 311–343.
8. Hanbery, J. W., and Dugger, G. S. Perithelial sarcoma of the brain: A clinicopathological study of thirteen cases. *A.M.A. Arch. Neurol. & Psychiat.*, 1954, *71*, 732–761.
9. Hsü, Y. K. Primary intracranial sarcomas. *Arch. Neurol. & Psychiat.*, 1940, *43*, 901–924.
10. Kane, C. A., and Foley, J. M. Primary reticulum-cell sarcoma of the leptomeninges. *A.M.A. Arch. Int. Med.*, 1958, *101*, 333–338.
11. Kehler, W. H., and Beck, E. Cerebellar sarcoma with bone metastases. *Radiology*, 1954, *63*, 736–740.
12. Kernohan, J. W., and Uihlein, A. Personal communication to the author.
13. Kinney, T. D., and Adams, R. D. Reticulum cell sarcoma of the brain. *Arch. Neurol. & Psychiat.*, 1943, *50*, 552–564.
14. Liu, C. T., and Selbach, G. Primary sarcoma of the leptomeninges: Report of three cases. *J.*

Neuropath. & Exper. Neurol., 1953, *12*, 186–193.

15. Lorenz, R. Differentialdiagnose der arteriographisch darstellbaren, intrakraniellen Geschwülste: Glioblastom, Meningeom, Sarkom. *Zentralbl. neurochir.*, 1940, *5*, 30–60.
16. McNeil, C. Hemangiopericytoma of the cerebellum. *Cancer Sem.*, 1957, *2*, 77–79.
17. Milletti, M. Die Differentialdiagnose der Gehirngeschwülste durch die Arteriographie. *Acta neurochir.*, 1950, Suppl. 1, pp. 1–79.
18. Nichols, P., Jr., and Wagner, J. A. Primary intracranial sarcoma: Report of nine cases with suggested classification. *J. Neuropath. & Exper. Neurol.*, 1952, *11*, 215–234.
19. Pendergrass, E. P., Schaeffer, J. P., and Hodes, P. J. The Head and Neck in Roentgen Diagnosis. Second edition, Charles C Thomas, Publisher, Springfield, 1956.
20. Rottino, A., and Poppiti, R. Diffuse meningeal sarcoma. *J. Neuropath. & Exper. Neurol.*, 1943, *2*, 190–196.
21. Russell, D. S., Marshall, A. H. E., and Smith, F. B. Microgliomatosis: A form of reticulosis affecting the brain. *Brain*, 1948, *71*, 1–15.
22. Seitz, D., and Kalm, H. Zur Diagnose der primären Hirnsarkome. *Deutsche Ztschr. Nervenh.*, 1958, *177*, 597–617.
23. Sparling, H. J., Jr., and Adams, R. D. Primary Hodgkin's sarcoma of the brain. *Arch. Path.*, 1946, *42*, 338–344.
24. Sparling, H. J., Jr., Adams, R. D., and Parker, F., Jr. Involvement of the nervous system by malignant lymphoma. *Medicine*, 1947, *26*, 285–332.
25. Stout, A. P. Hemangiopericytoma: A study of twenty-five new cases. *Cancer*, 1949, *2*, 1027–1054.
26. Troland, C. E., Sahyoum, P. F., and Mandeville, F. B. Primary mesenchymal tumors of the brain, so-called reticulum cell sarcoma: A report of five cases. *J. Neuropath. & Exper. Neurol.*, 1950, *9*, 322–334.
27. Yuile, C. L. Case of primary reticulum cell sarcoma of the brain: Relationship of microglia cells to histiocytes. *Arch. Path.*, 1938, *26*, 1036–1044.

THE NORMAL BRACHIAL CEREBRAL ANGIOGRAM

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ACCURATE evaluation and adequate medical management of patients manifesting a variety of types of cerebral dysfunction are coming to depend increasingly upon special diagnostic techniques. Angiography is the most recent and promising of these methods. There is no longer doubt that pre-angiographic clinical neurologic diagnoses, even when carefully arrived at, often err seriously in failing to identify specific provocative lesions which have produced the clinical state.[8] These lesions frequently are disclosed by cerebral angiography. During the past few years, roentgenologic study of the patterns of opacification in the cerebral and cervical circulatory tree has, therefore, become indispensable in the diagnostic armamentarium of the neurologist and the neurologic surgeon.

In view of the unreliability of clinical symptoms and signs in "stroke" syndromes,[7] there is a growing body of scientific opinion maintaining that each patient who has had a cerebrovascular accident deserves the benefit of complete cerebral angiography.[10] Cerebral angiography is equally valuable in the determination of other causes of cerebral malfunction. It is the only present method capable of yielding a precise anatomic diagnosis in patients harboring cerebral aneurysms; its value in localization of brain tumors has been well established; and recently its diagnostic preeminence has been emphasized in the handling of patients with intracranial injuries.[3] Finally, it is well known that congenital variations in vessels making up the circle of Willis are common and may be extreme, although asymptomatic. Angiography alone will demonstrate these anomalies of cerebral circulation during life.

The above considerations support the belief that contrast roentgenographic study of the cerebral circulatory tree will become more common as its importance is generally recognized in establishing diagnosis for diverse cerebral malfunctions. In the light of new knowledge of the frequency of asymptomatic segmental arterial lesions in the general population, it has become urgently necessary that a safe, reliable angiographic technique be developed to visualize not only the cervical carotid bifurcations but the major thoracic trunks upon which the cerebral circulatory tree stands. There is increasing awareness of the key role played by collateral circulation in cerebral failure.

Under optimal conditions, cerebral angiography should opacify the entire cervical and intracranial circulation, demonstrating conditions of blood flow and distribution normal to that individual without producing vessel overlap that confuses roentgenographic interpretation. Determination of the total capacity of the cerebral circulatory tree including speed and patterns of the consecutive phases of arterial filling, capillary transition, and venous drainage would seem to be of considerable value. These ends must ideally be met under artifact-free conditions, without risk or unusual discomfort to the patient and, above all, without traumatizing the arterial system carrying blood to the brain.

Percutaneous techniques of cerebral angiography which are in use by most clinics today suffer from several major disadvantages. Trauma to the common or internal carotid arteries or to the vertebral artery is inherent in these blind techniques and cannot be avoided.[12] Sequential filling of the carotid-cerebral and vertebral-basilar circulations is not obtained under conditions simulating normal blood flow for that individual. Finally, puncture methods

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fail to provide artifact-free opacification of widely separated parts of the cerebral arterial and venous trees.

The roentgenographic picture provided by carotid or vertebral injection is fragmentary at best. In many clinics the usual cerebral artery survey constitutes percutaneous carotid puncture on the side of the suspected cerebral dysfunction, with single angiograms taken in the occipital and lateral projections at the time of each injection. In some hospitals a desire for more complete arterial studies in patients with hemorrhagic[13] and ischemic cerebral dysfunction has led to adoption of routine bilateral carotid angiography. Despite these two percutaneous punctures, the vertebral basilar system remains an unknown, and the status of the origins of vertebral and common carotid arteries is in doubt unless additional injections are carried out. If serial angiograms have not been obtained, judgment in regard to roentgenographic abnormality rests upon interpretation of one to three films. Unfortunately, these may have been timed too early to identify a late filling cerebral lesion or too late to diagnose disease in proximal parts of the cerebral circulation.

The present communication describes an angiographic technique which (1) reliably opacifies two thirds of the cerebral circulation, from its sources to collecting veins, with each injection of opaque medium, (2) eliminates the trauma accompanying percutaneous puncture of arteries and (3) permits accurate and comfortable positioning of the patient. The vertebral-basilar system is regularly filled with contrast material under conditions approximating the physiologic normal for that individual. The common and internal carotid arteries are sharply opacified throughout their courses. Roentgenologic visualization of ascending and descending segments of the cerebral circulatory tree is obtained using a film changing device.

The diagrams in Figure 4 illustrate the normal vascular anatomy seen in sequential anteroposterior and lateral projections of an adult and child during the course of brachial cerebral angiography. Certain deviations from normal arterial patterns are pointed out. There has been no morbidity and no mortality accompanying this procedure in a series of over 200 adults and children.

TECHNIQUE

Brachial cerebral angiography is performed in the Radiology Department. Facilities are present to permit safe application of surgical technique. The team consists of an x-ray technician, surgical nurse, circulating nurse, and angiographer.

Premedication of the adult patient includes administration of parenteral phenobarbital and scopolamine one hour prior to the procedure. Occasionally, morphine sulfate is given on arrival of the patient at the angiography room so it will be exerting maximal effect at the time of injection of the opaque medium. Equipment utilized is shown in Figure 1.

The skin of the right arm is painted with surgical preparation solutions; a sterile sleeve with cut-out hole is rolled up to the shoulder; and sheet draping of the patient is carried out. The site of incision is along the bicipital groove, in the upper part of the arm, usually where the pulsation of the brachial artery is most superficial. After linear infiltration of the skin with nesacaine* solution is performed, a 3 cm. incision is made, followed by blunt and sharp dissection of underlying fatty tissue. Two small rake retractors are inserted; the fascia of the intermuscular septum is identified and split; and the neurovascular bundle is carefully exposed. The brachial artery, which lies just beneath the slack median nerve, is isolated, lifted on two narrow rubber tapes, and cleaned of connective tissue for a short distance. Distal blood flow is occluded by tightening the tape and a nick is made in the artery parallel to its long axis. Under direct vision, a 12 gauge

* Trade name for chloroprocaine hydrochloride.

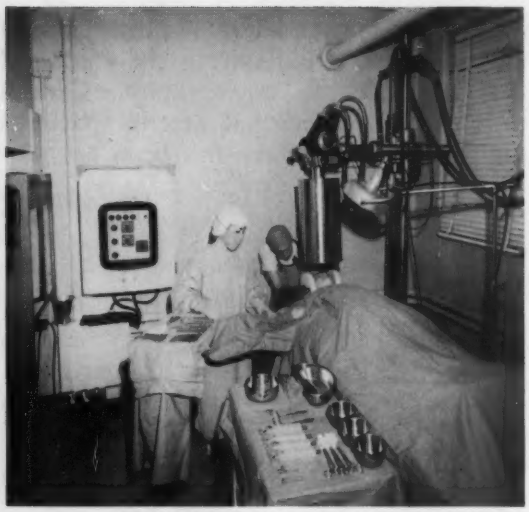

Fig. 1. Standard equipment for retrograde brachial angiography. The arm of the patient is abducted and supported on a board. Control panel for speed settings is at the left. The tube is set for an occipital series.

cannula 8 cm. in length with a solid, blunt stylet is inserted into the artery in the direction of the heart. A stopcock is situated midway along the cannula, and the stylet is tapered so that after it has been partially withdrawn the cock can be closed and the withdrawal completed without blood loss.*

Sterile saline irrigation is carried out through the cannula to make certain that free retrograde flow is present. After the patient has been warned to expect slight burning and aching in the arm, a 1 cc. test dose of 50 per cent hypaque† solution is injected and washed in with saline. The patient is observed over the next few minutes, while final preparations are being made for the initial angiographic series. To date, no anaphylactic or allergic reactions to this medium have been observed, and it is probable that in the future this precautionary testing can be discontinued.

The first series of films are exposed with the head in the occipital position. Considerable care is taken to include structures in the apex of the thorax. Depending upon the anatomy of the individual, the degree of tube obliquity may have to be modified slightly from patient to patient to obtain maximal upper thoracic visualization. Since there is no need for concern over dislodging a needle inserted in the neck, positioning can be carried out deliberately and with great accuracy. The head is immobilized by a technician who is adequately protected with gloves and apron.

Thirty cc. of 50 per cent hypaque is injected by the angiographer as rapidly as possible. A special thin-walled syringe with finger grips‡ enables the entire bolus of the opaque medium to be injected within less than a second. Serial exposure of the films is begun just as the plunger starts in. The syringe is immediately disconnected and the artery is flushed several times with 50 cc. volumes of saline solution. This irrigation seems to lessen the discomfort experienced mainly in the arm and shoulder. The patient is turned partially on his right side (a large sandbag beneath the left shoulder is satisfactory) and his head is rotated slightly to the lateral position. Injection of the second 30 cc. of opaque medium is accomplished in a manner identical to the first.

The single plane Elema-Schönander film changer, which has been used with this technique, permits a wide choice of speed settings. Sequences can be varied; speeds up to 6 frames per second can be obtained, or a pause may be interspersed in the series. A timing sequence frequently used has been three films in the first second, two in the second, a three to four second pause, and three films taken at intervals of one and one half seconds.

Films are developed immediately, scrutinized while wet, and judged to be technically satisfactory before the cannula is removed from the artery. The artery is reelevated on the tapes; a suture of 6-0 arterial silk is used to approximate the arteriotomy; and the vessel is replaced in its bed. Firm finger pressure on the line of closure for several minutes controls further

* Manufactured by Becton-Dickinson, Rutherford, New Jersey.

† Manufactured by Winthrop-Stearns, New York.

‡ Manufactured by Becton-Dickinson, Rutherford, New Jersey.

bleeding. Several subcutaneous and a locking skin suture are used to close the incision. The average time for artery cannulation is twenty minutes and the entire procedure is usually completed within an hour.

None of the patients have shown evidence of impaired circulation in the limb following brachial angiography. It is common for the radial pulse to be weak or unobtainable for several days, but it reappears thereafter in good strength. An occasional patient early in the series complained of slight paresthesias in the hand during the immediate postangiography period. These symptoms were thereafter easily prevented by taking particular care to avoid operative handling of the median nerve and are not likely to occur if the limb is positioned with slight flexion at the elbow.

DISCUSSION

In 1951 Radner,[11] impressed by the difficulty and dangers attending percutaneous vertebral angiography, advocated that arteriograms of this part of the cerebral system be obtained by direct catheterization of the vessel from the surgically exposed right brachial artery. His classic work included 221 patients studied by this technique. He apparently made no attempt to inject opaque material into the brachial, subclavian, or common carotid arteries. Retrograde injection of contrast substance into the right brachial artery for purposes of studying cerebral dysfunction was first reported by Gould *et al.*,[6] who recommended the method for showing the vertebral-basilar circulation in chlidren. In 1957 Collins *et al.*[4] applied the method to 17 adults, again reporting good filling of the vertebral-basilar complex following brachial arteriotomy and retrograde injection. In the course of applying this technique to adults, the present author observed in early cases that filling of *all* branches of the innominate artery would occasionally take place. It was soon discovered that rapid intra-arterial injection of a sufficient volume of contrast material resulted in opacification of the entire right cervical-cerebral vascular complex including the innominate, subclavian, common carotid and vertebral trunks and their peripheral branches. The method was easily perfected and has since been in routine use by the author.

Earlier studies and the present series have been entirely devoid of sequelae secondary to incision, catheterization, or the presently reported cannulation of the brachial artery. The danger of postoperative thrombosis or embolism is extremely unlikely, particularly if ordinary care is taken in the handling and postangiographic suturing of the artery. Since the brachial artery in the usual patient studied by this technique is unaffected by disease, the risk of producing an embolism in these patients due to the arteriotomy would appear to be far less than following iliac or femoral endarterectomy for occlusive segmental atherosclerosis. Yet this complication of thromboendarterectomy has not been reported, despite the frequency of such arterial surgery.

Successful restoration of blood flow through the region of brachial arteriotomy is always verified visually and by palpation before skin closure is begun. Even in the extraordinary event not yet encountered, such as rupture of the brachial artery, it is highly probable that few if any symptoms would follow ligation of the vessel. Blalock[2] and others have commented on the striking absence of symptoms after division of the vessel at any point along its course. Dangers attending brachial arteriotomy are, therefore, believed to be negligible in comparison with the known and unknown risks attending percutaneous carotid, vertebral, or subclavian puncture.[1]

Success in obtaining complete radiopaque delineation by retrograde brachial injection depends greatly upon rapid displacement of blood by the bolus of opaque medium. Delayed or intermittent injection results in escape of the opaque substance through the peripheral branches of the subclavian artery and especially in failure of the medium to reach the origin of the com-

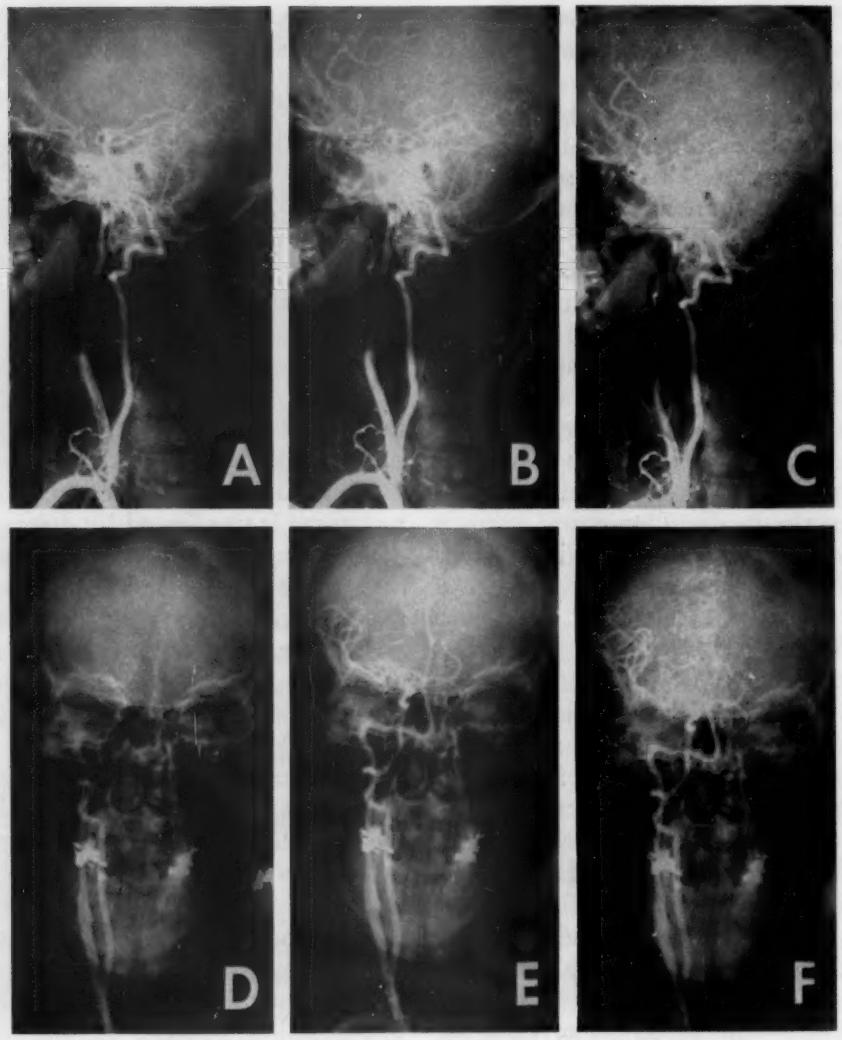

Fig. 2. (*A–F*) Retrograde angiograms of normal adult.

mon carotid artery. With a small volume of the opaque medium or slow injection, the bolus will travel centrally only as far as the mouth of the vertebral artery, resulting in opacification of that system alone. Mechanical advantages of a narrow bore, thin walled, positive pressure syringe allow the entire bolus of contrast material to be deposited intra-arterially in not more than a second.

Indications for the use of brachial cerebral angiography have been described elsewhere.[9] The technique is advocated in place of percutaneous artery puncture for patients in whom right carotid angiography is contemplated, for those requiring vertebral-basilar studies, and for patients undergoing survey of total cervical-cerebral circulation.

Initial filling occurs almost immediately under ordinary circumstances. Often, the first angiograms show opacification of the brachial, subclavian and innominate arteries as far proximally as the origin from the arch of the aorta. The system equilibrates with the injected volume of fluid and then direction of flow reverses, the medium escaping out of the branches of the innominate artery under normal arterial pressure relationships for that individual. The contrast material can be seen to fill thoracic and neck vessels during the first

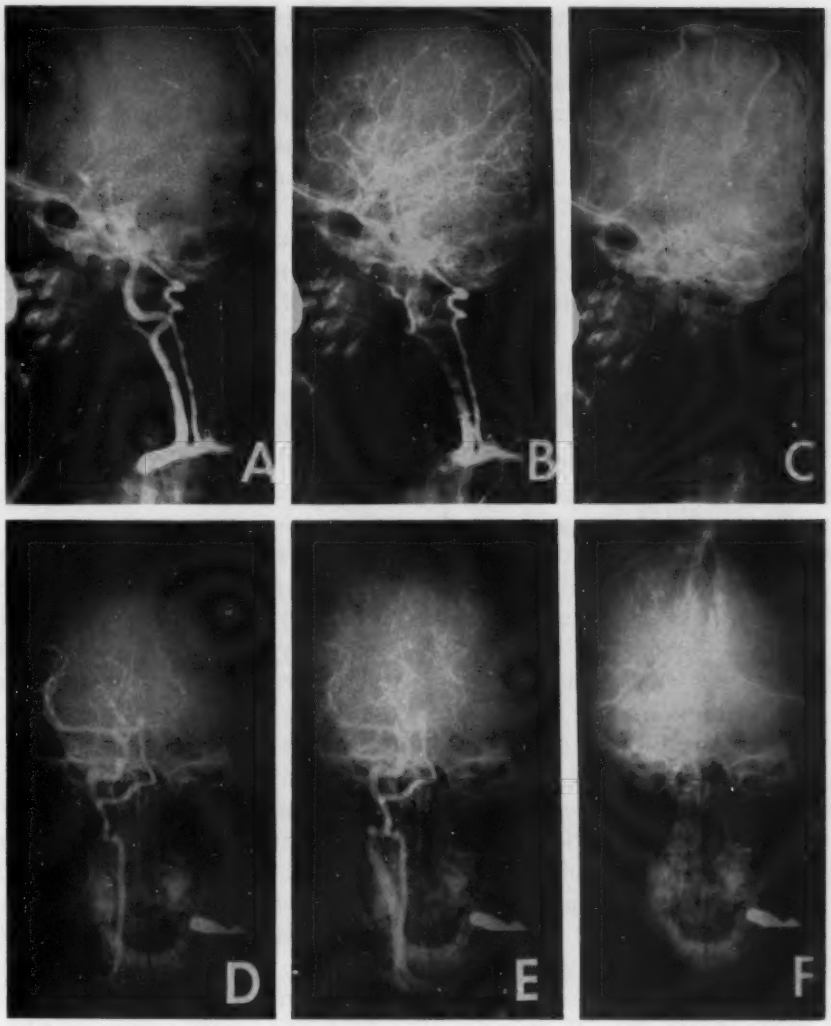

Fig. 3. (*A–F*) Retrograde angiograms of normal child.

two to three seconds (Fig. 2 and 3) and initial opacification of intracranial arteries occurs about three seconds after injection. Capillary phases normally occupy the third to sixth seconds and venous filling from the fifth to seventh. Speed of arterial flow in this system has been found to vary considerably with vascular or cerebral disease.

ROENTGENOGRAPHIC VASCULAR ANATOMY

In the standard anteroposterior or occipital projections, the subclavian artery can be seen curving centrally across the apex of the thorax. Arising from the artery, as it goes toward the heart, are the sites of origin of the transverse cervical, thyrocervical trunk, internal mammary, and vertebral arteries in that order.

Axillary Artery. Most of the axillary artery is visible, curving medially and superiorly. Below the shadow of the right clavicle and lateral to it, the large thoracoacromial trunk takes origin. Medially to this vessel, branches of the axillary are rarely seen. The course of the axillary artery is somewhat variable, depending on the position of the arm. With the limb in moderate abduction, the convex arc of the vessel is interrupted at the lower border of the first rib. A definite "kink" or reversal of

curve occurs normally at this junction with the third segment of the subclavian artery, and slight luminal narrowing is noted in many individuals. It is probable that fascial supporting layers anchor the artery as it crosses the costal surface. The axillary artery becomes the subclavian 8 to 10 cm. lateral to the midvertebral line.

Subclavian Artery. This large trunk is shaped like a broadened "C" with the open end toward the lung. From the lower border of the first rib, the subclavian inclines upward and medially at a 30 degree angle, crosses the shadow of the clavicle at the latter's midpoint and reaches its apex 4 to 6 cm. from the vertebral midline (Fig. 4). It then reverses its course abruptly, descending sharply toward the central sternum. On angiograms this curve reversal is seen to occur about 2.5 cm. medial to the midclavicular line. The subclavian terminates medially by merging with the common carotid to form the innominate artery.The subclavian rarely gives off branches lateral to its apex, although sometimes the thyrocervical trunk arises at the apex itself. In most instances, the lower surface of the subclavian arch is below the pleural apex and upper pole of the right lung, while in more than half of the populace the highest point of the artery is actually *below* the pleural apex.[5] In 3 out of 5 patients, the apex of the pleural cupula will, therefore, project above the level of the arch formed by the subclavian artery. These anatomic data indicate clearly the difficulty in performing percutaneous subclavian puncture without producing pulmonic injury.

Thyrocervical Trunk. This artery arises with surprising constancy from the superior surface of the subclavian arch, close to the apex (Fig. 4). The three radiating branches seen in textbooks are rarely distinguishable and the main trunk may be absent, individual branches originating directly from the subclavian. Transverse scapular and cervical trunks wind laterally, both above and below the subclavian and axillary arteries. The size and distribution of these branches vary widely from patient to patient, and no organizational pattern has been detected. According to their areas of distribution, the terminal branches of inferior thyroid, transverse scapular, and transverse cervical arteries can usually be distinguished.

Internal Mammary Artery. Approximately 1 cm. medial to the origin of the thyrocervical trunk, the internal mammary artery originates from the inferoposterior surface of the subclavian arch (Fig. 4). It runs slightly forward, laterally and down, to enter the thorax behind the cartilage of the first rib. This artery has been a constant finding in brachial angiography, particularly in regard to its site of origin.

Vertebral Artery. The vertebral artery is one of the most constant vessels arising from the subclavian. Branches are a rarity, although an occasional twig is seen to pass to the thyroid region. Relationships in the lower cervical region are best seen in occipital angiograms. The trunk emerges from the superior, slightly posterior surface of the subclavian artery, at the midpoint of its descending limb, 0.5 cm. medial to the origin of the internal mammary artery (Fig. 4). In the child, the vertebral artery is often practically equivalent in size to the common carotid artery, whereas in the adult the normal ratio is about 1 to 3 (Fig. 5, *A–D*).

The first portion of the vertebral artery is seen to ascend in a straight line for a distance of 3 to 6 cm.; then there is a gentle swing medially as it diverges to enter the transverse processes of the cervical vertebrae (Fig. 2, *A–F*). It is interesting that the point of this vertebral penetration by the artery varies considerably from patient to patient. The percutaneous technique of vertebral angiography utilizes as an anatomic landmark the presumed entrance of the artery into the transverse process of the sixth cervical vertebra, and needle puncture of the vessel is attempted at that location. On angiograms of a number of patients, the vertebral artery has been seen to ascend to the level of the fourth cervical vertebra before swinging to enter the foramen. This unpredictable variability in course would appear to explain some of the failures encoun-

FIG. 4. Diagrams of vascular anatomy opacified during brachial cerebral angiography of an adult (*above*) and a child (*below*).

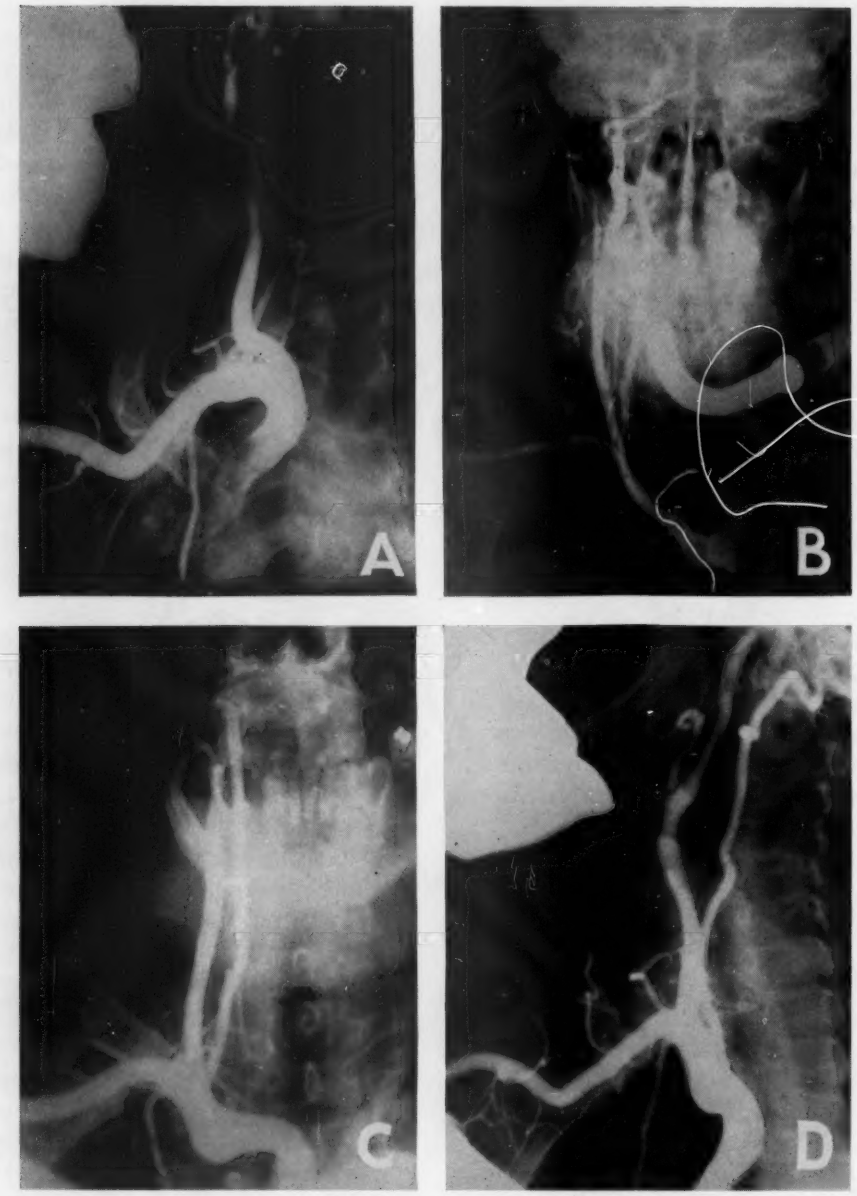

Fig. 5. The origins of the right cerebral vessels. Note the characteristic differences between (*A* and *B*) child and (*C* and *D*) adult patterns.

tered during percutaneous vertebral angiography.

After entering the foramina of the transverse processes, the vertebral artery ascends in a smooth, straight line to the level of the interspace between the body of the axis and the third cervical vertebra where it makes an abrupt right angle swing laterally for a distance of 1.5 cm. (Fig. 2, *A–F;* and 3, *A–F*). It then curves directly upward, inclining posteriorly at an angle of about 45° to pass through the foramen of the transverse process of the atlas. At the superior surface of the posterior arch, the artery once again executes a right angle swing, forming a very characteristic loop, then moves medially in a concave ascending arc to the suboccipital triangle. It

pierces the dura mater, inclining gradually toward the midline where it unites with the opposite vertebral artery.

Common Carotid Artery. This artery, equal in size to the subclavian, originates together with the latter as a paired structure from the innominate trunk about 2.5 cm. proximal to the subclavian apex. The common carotid emerges medial to the subclavian, and sometimes originates anterior to it (Fig. 4). It courses upward, curves laterally in the base of the neck to cross the vertically ascending vertebral artery at a slight angle, then runs directly upward and parallel to that vessel to bifurcate into the internal and external carotids at the level of the angle of the jaw.

The roentgenographic anatomy of the intracranial distribution of the internal carotid and vertebral arteries has been described in a number of texts. Both of these systems are filled with contrast medium simultaneously during certain sequences of the brachial injection technique, and slight confusion regarding identity of branches may be encountered when scanning occipital angiograms. Overlap of the right posterior cerebral and the right anterior cerebral arteries is sometimes puzzling. These arteries are sharply defined and easily distinguished, however, in the lateral view.

Flow patterns in the circle of Willis normal for that patient are seen particularly well in lateral views. The patency and functional adequacy of the homolateral posterior communicating artery, which is so frequently the site of congenital malformation, can be accurately assessed. Opportunity is presented for study of collateral cerebral circulation under conditions much more close to a physiologic norm, since pressure artifacts impossible to dissociate from percutaneous techniques are not present during retrograde angiography.

SUMMARY

1. Retrograde brachial angiography reliably opacifies two thirds of the cerebral circulation without injuring any part of that system.

2. The technique of this procedure is described and a representative series of normal angiograms is presented for the adult and child.

3. Based on more than 200 brachial angiograms, certain roentgenographic features of the upper thoracic circulation are discussed.

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REFERENCES

1. Abbott, K. H., Gay, J. R., and Goodall, R. J. Clinical complications of cerebral angiography. *J. Neurosurg.*, 1952, *9*, 258–274.
2. Blalock, A. Study of surgical treatment of congenital pulmonic stenosis. *Ann. Surg.*, 1946, *124*, 879–887.
3. Carton, C. A. Cerebral Angiography in the Management of Head Trauma. Charles C Thomas, Publisher, Springfield, Ill., 1959.
4. Collins, W. F., Jr., Slade, H. W., and Lockhart, W. G. Brachial vertebral angiography in adults. *J. Neurosurg.*, 1957, *14*, 466–468.
5. Daseler, E. H., and Anson, B. J. Surgical anatomy of subclavian artery and its branches. *Surg., Gynec. & Obst.*, 1959, *108*, 149–174.
6. Gould, P. L., Peyton, W. T., and French, L. A. Vertebral angiography by retrograde injection of brachial artery. *J. Neurosurg.*, 1955, *12*, 369–374.
7. Gurdjian, E. S., Webster, J. E., Hardy, W. G., and Lindner, D. Surgical considerations in management of cerebrovascular disease. *Minnesota Med.*, 1958, *41*, 327–331.
8. Kuhn, R. A. Importance of accurate diagnosis by cerebral angiography in cases of "stroke." *J.A.M.A.*, 1959, *169*, 1867–1875.
9. Kuhn, R. A. Brachial-cerebral angiography. *J. Neurosurg.* In press.
10. Murphey, F., and Miller, J. H. Carotid insufficiency—diagnosis and surgical treatment; report of twenty-one cases. *J. Neurosurg.*, 1959, *16*, 1–23.
11. Radner, S. Vertebral angiography by catheterization; new method employed in 221 cases. *Acta radiol.*, 1951, suppl. 87, 1–134.
12. Rimpau, A., and Seils, H. Pathologisch-anatomische Befunde an der Punktionsstelle bei der Hirnarteriographie und Betrachtungen zur Punktionstechnik. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1957, *87*, 191–199.
13. Silver, M. L. Management of intracranial bleeding. *J.A.M.A.*, 1957, *163*, 1097–1102.

MEASUREMENT OF THE ANTERIOR CEREBRAL ARTERY IN NORMAL AND ABNORMAL CEREBRAL ARTERIOGRAMS*

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CEREBRAL angiography is the most frequently used roentgenographic technique in the detection of intracranial space occupying lesions. The normal cerebral angiogram has been well documented. An appreciation of the normal anatomy and variations of the intracranial circulation is a prerequisite for the accurate interpretation of cerebral angiograms. An intracranial space occupying lesion may result in a variety of changes seen on the cerebral angiogram. Many of these changes are quite evident and an accurate interpretation can be made. However, some of the lesser changes, especially a slight shift in the position of the vessels, may present considerable difficulty in interpretation inasmuch as the change from the normal may be minimal if not visually unappreciable.

Carotid cerebral angiography generally results in the visualization of the anterior and middle cerebral arteries and, less frequently, the posterior cerebral artery. Attempts have already been made to measure the course of the middle cerebral artery and its branches.[4,5] Although these measurements may be quite useful, the borderline between normal and abnormal cannot always be strictly defined.

Recently there have been similar studies of the anterior cerebral artery. Hodes *et al.*[2] have emphasized the possible wavering course of the anterior cerebral artery in the sagittal plane. This vessel has been studied in the lateral projection by Morris and Peck.[3] After dividing the anterior cerebral artery into four segments, they described the types and frequency of normal variations observed in each segment. These workers recorded the most numerous variations in the segment which encloses the genu of the corpus callosum. Variations in the course of the anterior cerebral artery were also described by Curry and Culbreth.[1] They recorded the variations and position of the anterior cerebral artery as it rounds the genu of the corpus callosum. Their measurements from the inner table of the skull in the anterior fossa to the genu curve of the anterior cerebral artery range from 1.5 to 2.5 cm. These authors concluded that the location of the anterior cerebral artery is an unreliable landmark for detecting the exact position of the genu of the corpus callosum. Furthermore, the pericallosal artery, although running superiorly to the corpus callosum, does not follow a fixed course and may often lie as much as 1.0 cm. above this structure.

Despite the previous discouraging results with mensuration of the anterior cerebral artery, a study was undertaken of the anterior cerebral artery in the normal and abnormal cerebral angiogram. In view of the apparently fixed site of the genu of the corpus callosum, attention was again concentrated on the segment of the anterior cerebral artery which curves around the genu. A statistical analysis of the results of this work is the basis of this report.

TECHNIQUE

The mensuration of the anterior cerebral artery was carried out at two separate institutions. Each team consisted of two radiologists. After deciding upon the lines to be

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measured, each group provided a series of normal and abnormal cerebral angiograms. Cross checks on measurements showed a very close reproduction of results. The following eight measurements were originally used in the study:

Measurement 1. A point is determined on the pericallosal artery, 1 cm. posterior to the highest point on the genu of the corpus callosum. From this point a line is drawn perpendicular to the inner table of the skull.

Measurement 2. A line is drawn from the most anterior point of the floor of the anterior fossa (posterior wall of the frontal sinus) to the point on the genu closest to the anterior cerebral artery.

Measurement 3. A line is drawn from the identical point on the genu curve of the anterior cerebral artery, as in measurement 2, to the lambda, which is determined by a line 7 cm. in length above the internal occipital protuberance along the inner table.

Measurement 4. This is a line from the tuberculum sellae to point 1 on the anterior cerebral artery.

Measurement 5. This involves determination of the position of the anterior cerebral artery in the sagittal plane and a measurement of the amount of shift beyond the midline in the abnormal cerebral angiogram.

Measurement 6. This is measurement of the length of the skull from the floor of the anterior fossa anteriorly to the internal occipital protuberance posteriorly.

Measurement 7. The height of the skull, as determined by a line 8 cm. above the anterior fossa on the inner table of the skull drawn to the tuberculum sellae, is measured.

Measurement 8. The height of the skull, as determined by a line drawn perpendicularly from the tuberculum sellae to line 6 and extended through to the inner table of the skull.

It will be observed that lines 1, 2, 3, and 4 are related to the anterior cerebral artery. Line 5 is a measurement of the degree of shift of the anterior cerebral artery due to any space-occupying lesion. Lines 6, 7 and 8 were used to determine possible relationships of the first four lines to the skull configuration, specifically, height and length.

RESULTS

The eight linear measurements were applied to a group of 97 apparently normal cerebral angiograms as well as to a group of 27 angiograms demonstrating confirmed space occupying lesions in the frontal lobe.

The standards for measurements of the lateral cerebral angiogram were lateral studies performed with a Fairchild or Sanchez Perez serialographic unit.

At one of the institutions taking part in this study, more recently a Biplane Schönander unit has been used for cerebral angiography. The resulting roentgenograms show some magnification of the skull in the range of about 20 per cent. On such cerebral angiograms, a correction factor was determined based on the conventional lateral roentgenogram of the skull.

The normal and abnormal cerebral angiographic measurements were then statistically analyzed. The abnormal cerebral angiograms were divided into three separate groups depending upon the location of the space occupying lesion in the frontal area: A. anterior to the genu of the corpus callosum (left abnormal); B. above the genu of the corpus callosum (above abnormal); and C. below and posterior to the genu of the corpus callosum (inside abnormal).

A statistical analysis was finally limited to six linear measurements: lines 1, 2, 3, 4, 6, and 7. The analysis of these six lines gave results which indicated that only lines 1 and 2 might be useful in differentiating the normal from the abnormal cerebral angiogram (Fig. 1). There was no correlation between lines 1 and 2 and the configuration of the skull (lines 6, 7, and 8—height and length).

For purposes of statistical evaluation these two linear measurements were each considered in the following manner: *Line 1.* Normal cerebral angiogram: a. group vari-

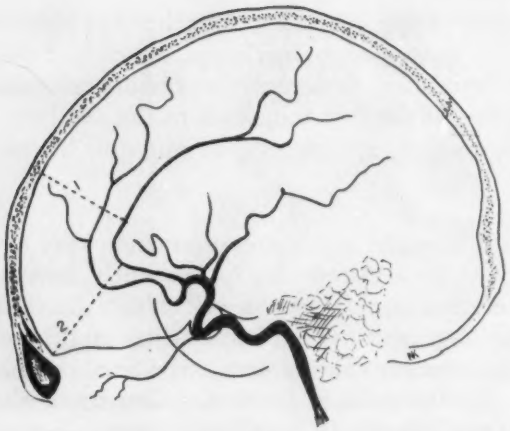

Fig. 1. Line drawing of lateral cerebral arteriogram. *Lines 1* and *2* are indicated. See test for reference points used to locate lines.

ation; b. individual variation. *Line 2*. Abnormal cerebral angiogram (Groups A, B, and C): a. group variation; b. individual variation.

All of the aforementioned statistical data indicate range and differences at a probability level of 1 per cent.

The results of measurement 1 of individual and group variation between the normal and abnormal cerebral angiograms are indicated in Figures 2 and 3. In the case of individual variation, it can be seen that the normal measurement 1 includes measurements in the three abnormal groups. The inside abnormal group shows slight extension beyond the lower limits of the normal and the left abnormal group shows extension beyond the upper and lower limits of the normal. When measurement 1 is considered as a group variation, as indicated in Figure 3, greater separation is apparent between the normal and the groups with abnormality above and inside. There is, however, some overlap in the group marked left abnormal.

The results for measurement 2 are indicated in Figures 4 and 5. Comparison for individual variation shows overlap between the normal and the three abnormal groups, although the three abnormal groups have upper limits which extend beyond the normal variation. Again, more distinction is possible when groups, rather than individuals are considered in the normal and abnormal cerebral angiograms.

DISCUSSION

The results of this study indicate that it is possible to have two linear measurements which singly or combined may be used to determine the normality or abnormality of

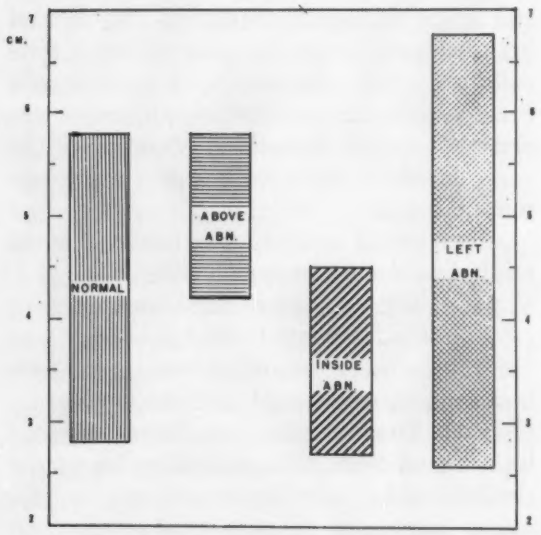

Fig. 2. Comparison of individual variation of measurement 1 in normal and abnormal arteriograms.

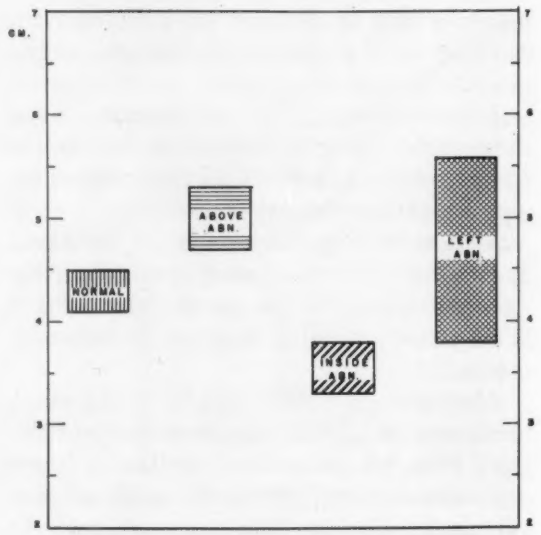

Fig. 3. Comparison of group variation of measurement 1 in normal and abnormal arteriograms.

a cerebral angiogram. Unfortunately, the extremes of normal and abnormal are such that clear-cut delineation between normal and abnormal is not possible in an individual case. Groups of cases are thus statistically more reliable, as indicated by this study, in determining the normality or abnormality of a given measurement. In everyday work, linear measurement 1 is probably most valuable. This measurement not only reflects the position of the corpus callosum, but also may be used to measure an upward or downward shift of the pericallosal artery by a space occupying lesion. In addition, this vessel reflects the size of the underlying lateral ventricle. When the ventricles dilate, as occurs in hydrocephalus of the communicating or noncommunicating type, the enlarged ventricle may elevate the pericallosal artery. However, the elevation of the pericallosal artery probably does not reflect the true size of the lateral ventricles inasmuch as, when the lateral ventricles dilate, the pericallosal artery may slide towards the medial portion of the ventricle and thus not lie on the roof of the ventricle. Thus, a line 1 which is shorter than average, in the absence of other signs of a space occupying lesion, would suggest a dilated ventricle.

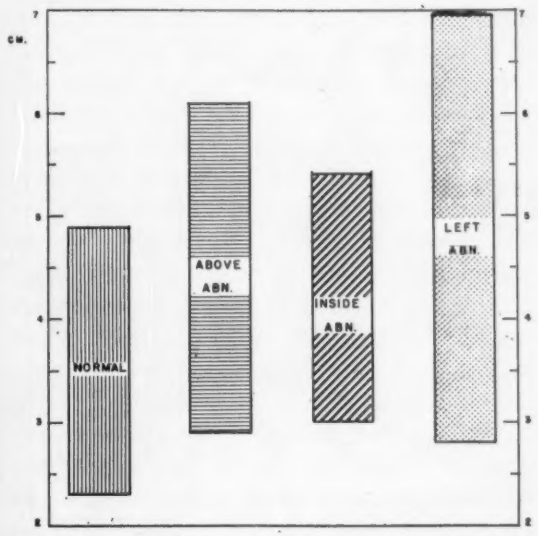

Fig. 4. Comparison of individual variation of measurement 2 in normal and abnormal arteriograms.

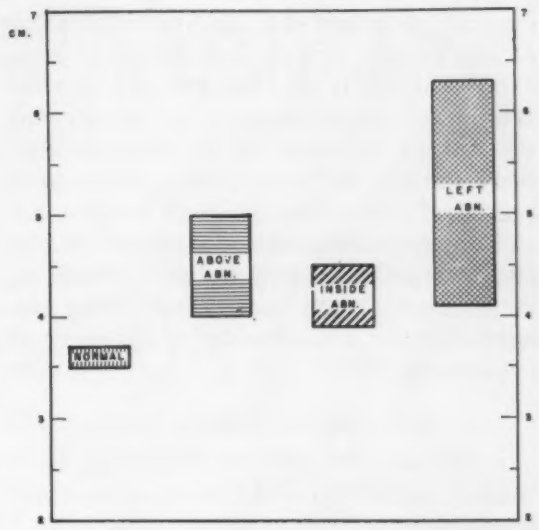

Fig. 5. Comparison of group variation of measurement 2 in normal and abnormal arteriograms.

Although many of the abnormal cerebral angiograms upon which these measurements were applied were quite obviously abnormal to the usual visual interpretation, there were other instances in which the findings were not as striking and the measurements could then be applied for confirmation of abnormality.

One must appreciate the difficulty encountered in this study in placing the space occupying lesion into one of the three abnormal groups already described. These mass lesions are not necessarily sharply delineated and may exert a three dimensional effect on the surrounding cerebral vessels. The exact localization, size and growth characteristics of the mass determine the pattern of vascular displacement. Thus, a space-occupying lesion may cause a change in both measurements 1 and 2. The change may be in the same direction (increase in 2 and decrease in 1 or vice versa). The failure of linear measurements to completely differentiate the normal and abnormal cases confirms an old observation in medicine that there is a range of the normal which overlaps the abnormal. The three dimensional aspect of intracranial space oc-

cupying processes is an additional limiting factor. Finally, this study indicates at present that there is no fixed position or relationship of the anterior cerebral artery and the corpus callosum to the surrounding bony structures. Nevertheless, the results presented define the limits of normal for two linear measurements applied to the anterior cerebral artery and can, therefore, be of some assistance in establishing the normality or abnormality of a cerebral angiogram.

CONCLUSIONS

1. The anterior cerebral artery has been studied in normal and abnormal cerebral angiograms.

2. Two linear measurements applied to the anterior cerebral artery on the lateral angiogram are described.

3. Statistical analysis of the two linear measurements indicates that individual cases show too much variation and overlap to permit separation of the normal and abnormal angiograms. However, groups of normal and abnormal studies show less variation, permitting some useful application of these two measurements.

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REFERENCES

1. CURRY, R. W., and CULBRETH, G. G. Normal cerebral angiogram. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *65*, 345–373.
2. HODES, P. J., CAMPOY, F., RIGGS, H. E., and BLY, P. Cerebral angiography; fundamentals in anatomy and physiology. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1953, *70*, 61–82.
3. MORRIS, A. A., and PECK, C. M. Roentgenographic study of variation in normal anterior cerebral artery. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1955, *74*, 818–826.
4. TAVERAS, J. M., and POSER, C. M. Roentgenologic aspects of cerebral angiography in children. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1959, *82*, 371–391.
5. WORINGER, E., and GERNEZ, A. L'artériogramme cérébral: essai de définition des frontières de l'artériogramme carotidien normal et de ses variations. Analyse de 100 cas. *Presse méd.*, 1948, *56*, 881–882.

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A SIGN OF CEREBRAL VENTRICULAR DILATATION OBSERVED IN CAROTID PHLEBOGRAMS*

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CAROTID arteriograms in cases of internal hydrocephalus are characterized by changes in the arteries adjacent to the cerebral ventricles. The anterior cerebral artery is elevated in a smooth curve because of the corresponding rise of the corpus callosum, losing its normal undulations and following a smooth curve which is convex anteriorly. The posterior portions of the branches of the anterior cerebral artery run almost parallel to the middle cerebral artery. The middle cerebral artery, which lies above the temporal horn of the lateral ventricle, is elevated and displaced forward so that its angle with the base of the skull is increased (Egas Moniz, 1940[1]). In addition, there is moderate or marked stretching of the blood vessels because of the associated increased intracranial pressure.

The degree of convexity of the curve of the anterior cerebral artery and the upward and forward displacement of the sylvian arterial group, however, are not proportionate to the degree of ventricular dilatation, so that, when an estimation of the actual size of the hydrocephalic ventricle is desired, direct visualization by means of contrast filling of the ventricular system is necessary.

As cerebral angiography became more widely used as an adjunct in neuroradiologic diagnosis, the present authors paid special attention to the findings on cerebral angiograms in cases of internal hydrocephalus, with the possibility in mind of directly demonstrating the actual size of the ventricle. As a first step, the veins on the wall of the lateral ventricle in cerebral phlebograms were considered. It was felt that the length of these veins on the surface of the lateral ventricle might be in proportion to the actual size of the lateral ventricle. The veins involved here are the vein of the septum pellucidum and the vein of the thalamostriatum and its tributaries, branching upon the caudate nucleus and flowing into the internal cerebral vein.

TECHNIQUE AND OBSERVATIONS OF A SIGN OF VENTRICULAR DILATATION

The usual angiographic procedure with percutaneous carotid puncture and an injection of about 10 ml. of 60–76 per cent of urografin (Schering, Berlin) was performed under intravenous anesthesia. Phlebograms were made 6.0–6.5 seconds after arteriography. These phlebograms proved satisfactory in demonstrating the deep venous system of the cerebrum. It was observed that in the lateral phlebograms the peripheral ends of the well visualized veins on the inner surface of the lateral ventricle showed a T form and that the horizontal arms of the T presented as a smooth contour outlining the anterior and parietal parts of the lateral ventricle. Figure 1 shows the normal ventricular contour while the slightly, moderately and markedly dilated ventricular contours which were observed are illustrated in Figures 2 to 4. In cases where moderately or markedly dilated ventricles were noted, direct contrast filling of the ventricular system was also carried out in order to confirm the phlebographic findings. In the presence of internal hydrocephalus, the veins on the surface of the lateral ventricle were elongated in proportion to the dilatation of the lateral ventricle; for instance, in markedly hydrocephalic patients, the vein of the septum pellucidum and the vein of the thalamostriatum were so long that their

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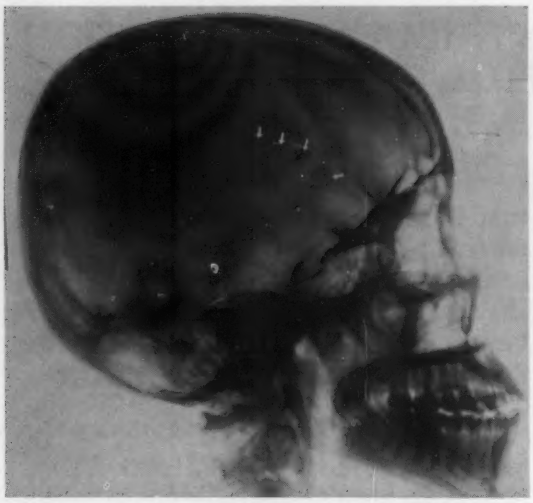

FIG. 1. Phlebogram showing the normal ventricular contour.

FIG. 3. Phlebogram showing the moderately dilated lateral ventricle in a patient having acoustic neurinoma.

peripheral ends were visualized near the superior sagittal sinus. In addition, the vein of the thalamostriatum branched off at a right angle from the internal cerebral vein, with stretching of the blood vessels.

Cerebral phlebography is limited in that only the anterior and parietal parts of the lateral ventricle are visualized and not the whole ventricular system; however, this appears to be adequate as a basis for estimating the degree of internal hydrocephalus, except when there is some deformity in the occipital or temporal horns of the lateral ventricle.

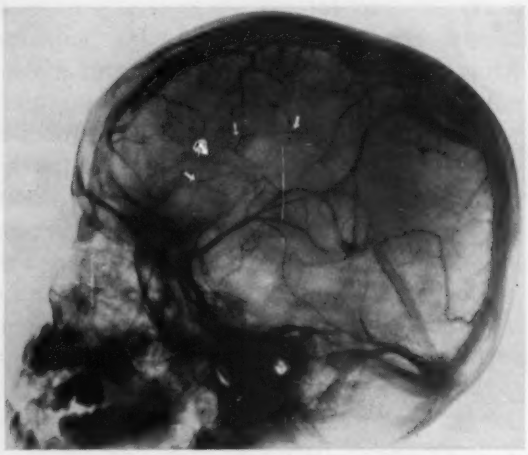

FIG. 2. Phlebogram showing the slightly dilated lateral ventricle in a patient having pontine glioblastoma.

SUMMARY

A characteristic contour of the lateral ventricle can be detected in cases of internal hydrocephalus by cerebral phlebography by the visualization of the veins running on the inner surface of the anterior

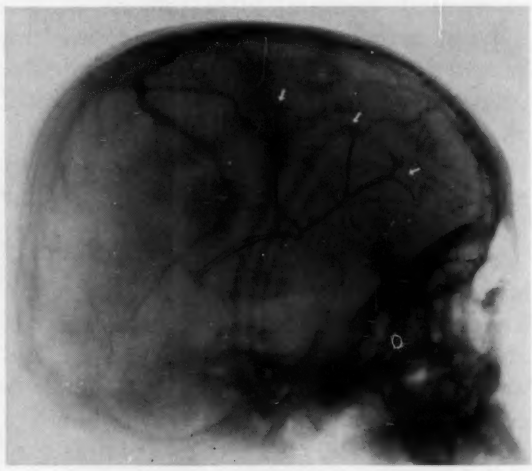

FIG. 4. Phlebogram showing the markedly dilated lateral ventricle in a patient having inflammatory aqueduct stenosis. Note the stretching and right angle branching of the vein of thalamostriatum and the severe elongation of the veins on the anterior and parietal wall of the lateral ventricle.

and parietal parts of the lateral ventricle. In cases of internal hydrocephalus, these veins (vein of the septum pellucidum, vein of the thalamostriatum, etc.) are stretched and elongated in proportion to the dilatation of the lateral ventricle and the vein of the thalamostriatum branches off at a right angle from the internal cerebral vein. Thus, in the lateral projection the upper margin of the anterior and parietal parts of the lateral ventricle is delineated by a convex curve formed by the peripheral ends of these veins.

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REFERENCE

1. Egas Moniz. Die cerebrale Arteriographie und Phlebographie. In: Handbuch der Neurologie. Bumke, O., and Foerster, O., Editors. Julius Springer, Berlin, 1940.

A METHOD FOR ROENTGENOLOGIC MEASUREMENTS FROM SPOT FLUOROGRAMS*

ITS APPLICATION TO SKULL CAPACITY DETERMINATION

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ROENTGENOGRAPHIC volume determinations *in vivo* of certain internal organs, for instance the heart, have proved to be of practical clinical importance.

In this JOURNAL, as well as elsewhere, reports have been published on roentgenographic methods applied to dry crania for determining the skull capacity, and the accuracy of the results has been analyzed. The present work is aimed at applying a similar method to living material and at comparing these results with previously obtained values. We also wish to introduce a modification of the technique by using smaller film size, *viz.*, photofluorography, in order to test the accuracy of this procedure. This involves smaller film costs and is more convenient for mass surveys. Finally, a practical and convenient method is proposed for correcting the reduction in size due to photofluorography.

PRINCIPLE OF METHOD

Haas,[3] in his roentgenologic investigation of live skulls, the capacities of which were not known, pointed out that skull length alone quite reliably characterizes the human skull capacity.

MacKinnon *et al.*,[4] in their study of human skulls of known volume, showed that the capacity of human skulls can more accurately be measured from two roentgenograms taken in lateral and anteroposterior projections.

The determination of skull capacity from two roentgenograms is based on a modified formula for the capacity of an ellipsoid:

$$V=(\tfrac{1}{2}\times L\times H\times W)+(\tfrac{1}{2}\times L\times B\times W)\times 0.51$$

where V = skull capacity, L = maximum internal anteroposterior diameter, H = height measured from midway between the projections of the external auditory meatuses to the farthest point on the inside vault of the skull, B = bregma posterior-cranial fossa diameter, and W = maximum internal breadth (Fig. 1, *A* and *B*).

Bergerhoff[1] has mathematically shown the reliability of the above formula.

In our study we used the left lateral and posteroanterior projections. The measurements of L, H, and B were obtained from the lateral roentgenogram, the width (W) from the posteroanterior roentgenogram.

The purpose of this investigation is: (1) to present a simple method which makes it possible to obtain measurements from spot fluorograms, (2) to show that spot fluorograms can be applied to skull volume determination, and (3) to study the skull volumes of live adults and compare the calculated capacities with those obtained from dry human skulls by MacKinnon *et al.*[4]

MATERIAL AND TECHNIQUE

Our material comprises 113 female patients in a hospital for the mentally ill. The ages varied from thirty-three to ninety years.

The spot fluorograms were made with the Odelca X-ray mirror camera (skull type 70-XIX-U); the size of the fluorograms was 70×70 mm.; the tube-screen distance was 100 cm.

One lateral and one posteroanterior fluorogram were made of each patient. Before taking the lateral fluorogram, a small lead ball was fixed with adhesive tape on the frontal region of the patient's head and another similar indicator on the occiput, both in the sagittal plane (Fig. 2*A*). Before the posteroanterior fluorogram was made, the same indicators were placed

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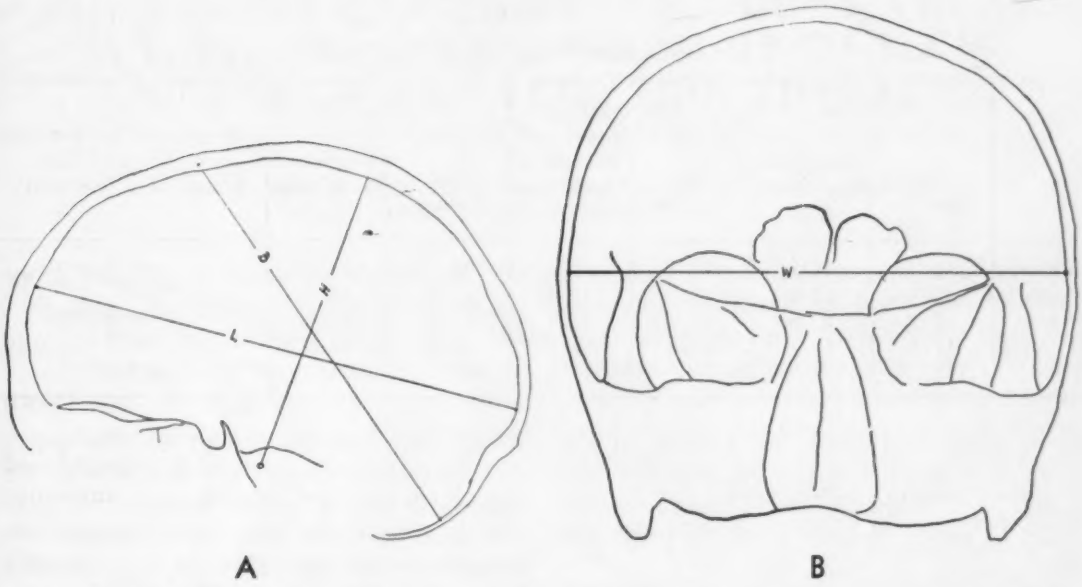

Fig. 1. Diagrams of (*A*) lateral and (*B*) anteroposterior roentgenograms showing measurements used in determining skull capacity. *L* = skull length, *H* = skull height, *B* = bregma measurement, *W* = maximum internal width. (Reproduced with permission from Georg Thieme Verlag. Bergerhoff, W., *Fortschr. a. d. Geb. d. Röntgenstrahlen.*[1])

on the parietal regions at the points of maximum external width of the skull (Fig. 2*B*). The distance between the indicators was measured with a caliper. During exposure the patient's head was fixed with a special support.

The fluorograms obtained were enlarged so that the distance between the indicators on the enlarged image was equal to that measured on the patient. We employed a conventional magnification apparatus used in photography. Since the indicators were placed on the patients in the same plane as that from which the measurements were

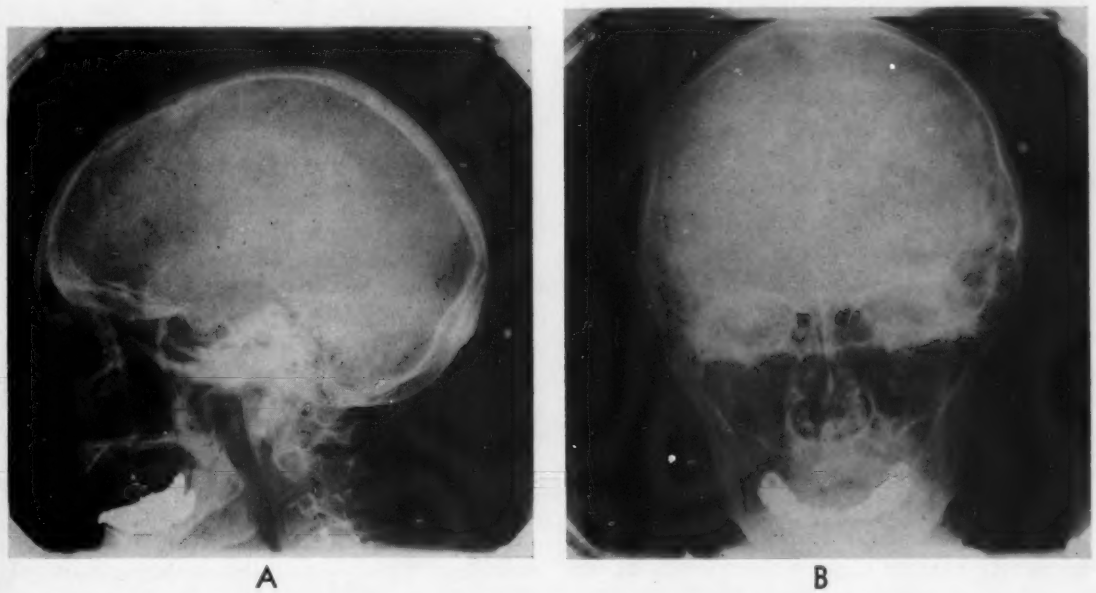

Fig. 2. (*A*) Lateral and (*B*) posteroanterior fluorograms showing the position of the lead indicators.

TABLE I

SKULL CAPACITY MEASUREMENTS

	Minimum		Maximum		Average		Difference in Our Averages from Those of MacKinnon *et al.* (per cent)
	Our Results	Results of MacKinnon *et al.*	Our Results	Results of MacKinnon *et al.*	Our Results	Results of MacKinnon *et al.*	
L (cm.)	14.7	15.25	17.8	18.2	16.3	16.6	−1.8
B (cm.)	12.5	13.0	15.3	15.45	14.0	14.0	±0.
H (cm.)	8.7	10.6	12.3	12.8	10.9	11.5	−5.2
W (cm.)	11.1	11.5	15.9	14.6	13.9	12.8	+8.6
V (cc.)	1,157	1,109	1,762	1,761	1,438	1,401	+2.6

to be made and since "all objects in the same film-parallel plane are equally distorted" (Büchner[2]), the measurements could be taken directly from the projected fluorograms.

RESULTS

Table 1 shows the minimum and maximum values of measurements in our material and the calculated averages. In addition, the corresponding results of MacKinnon *et al.*[4] are presented for comparison, as well as the percentile difference between the two studies.

The average skull volume in our material was 1,438 cc., which is only 2.6 per cent greater than in the material of MacKinnon *et al.* In MacKinnon's material of 52 skulls, an error of 2.4 per cent from the real volumes was observed in roentgenologically calculated skull capacities. Thus, the difference in the average skull volumes lies within the range of error inherent to the method.

In the linear measurements, the difference between our average values and those of MacKinnon *et al.* ranged from −1.8 to +8.6 per cent. The greatest deviation (+8.6 per cent) was observed in the width of the skull. Since the sagittal plane is an anatomically determined plane, it is easy to put the indicators on the patient so that they really lie on that plane. The widest part of the skull, however, is so formed that it is difficult to find by palpation any anatomically clearly identifiable points of orientation on which to place the indicators. In addition, the maximum *external* width may not always exactly coincide with the maximum *internal* width. Since the width of the skull was the only measurement not taken from the sagittal plane, a greater difference is to be expected.

SUMMARY

A practical and convenient method is proposed for correcting the reduction in size due to photofluorography. One hundred and thirteen skull capacities were measured from biplane spot fluorograms. An analysis of the accuracy of the method and comparisons of the results on material *in vivo* with earlier findings on dry crania are presented.

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REFERENCES

1. BERGERHOFF, W. Über die Bestimmung der Schädelkapazität aus dem Röntgenbild. *Fortschr. a.d. Geb. d. Röntgenstrahlen*, 1957, *87*, 176–184.
2. BÜCHNER, H. Orthodiametrie; die Grössenbestimmung mittels einfacher Röntgendurchleuchtung. *Fortschr. a.d. Geb. d. Röntgenstrahlen*, 1951, *74*, 498–511.
3. HAAS, L. L. Roentgenological skull measurements and their diagnostic applications. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1952, *67*, 197–209.
4. MACKINNON, I. L., KENNEDY, J. A., and DAVIES, T. V. Estimation of skull capacity from roentgenologic measurements. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *76*, 303–310.

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THE EXTENT OF INTRACRANIAL GLIOMATA AT AUTOPSY AND ITS RELATIONSHIP TO TECHNIQUES USED IN RADIATION THERAPY OF BRAIN TUMORS*

By JOSEPH P. CONCANNON, M.D.,† SIMON KRAMER, M.D.,‡ *and* RICHARD BERRY, M.D.§

THE value of radiation therapy in the treatment of intracranial gliomata has not yet been established. Reports in the literature are conflicting. Some[3,6] stress the usefulness of radiation therapy, while others[2] are less enthusiastic. Penman and Smith[5] pointed out that there has been no controlled investigation in this field, and concluded "we do not believe that radiation therapy of intracranial gliomata employed as an adjunct to surgery is useless any more than we believe it is useful. We merely find no satisfactory evidence for either belief."

A controlled investigation was therefore started in 1953 under the auspices of the Medical Research Council of Great Britain to determine the value of radiation therapy for patients with intracranial gliomata. The plan of this investigation has been described by Penman[4] in detail. Briefly, one group of patients with intracranial gliomata was to be treated by the indicated surgical procedures only. Another matched group of patients was to have radiation therapy as well. All surgical procedures were to be performed by the two participating neurosurgeons, Mr. McKissock and Mr. Logue. All the histologic reports were to be made independently by three pathologists, Prof. Crawford, Prof. Blackwood and Dr. McMenemey. The surgery was to be done in the Neurosurgical Departments of St. George's Hospital, the National Hospital for Nervous Diseases, Queen Square, and the Maida Vale Hospital for Nervous Disease, London. The radiation therapy was to be given at the Royal Marsden Hospital, a plan of cooperation in treatment having been in operation between these hospitals since 1949. One of us (S.K.) initially developed the radiation therapy techniques employed and directed the treatment during the first two years of the investigation. The radiation therapy approach was based primarily on two factors: the histological grading of the tumor and the apparent extent of the tumor as determined by clinical, roentgenologic and operative findings. The plan for patients with tumors of apparently limited extent and low grade malignancy (gliomata Grades I and II) was to continue to use the supervoltage rotation technique started in 1950, if the volume to be irradiated was smaller than an 8 cm. sphere. A tumor dose in the range of 6,500 to 7,000 r was to be given to this volume in six to seven weeks. If the volume to be irradiated was in excess of an 8 cm. sphere, patients were to be treated through stationary fields, again using supervoltage apparatus wherever possible. The planned tumor dose was then to be in the range of 6,000 to 6,500 r in six to seven weeks. Patients with tumors of higher malignancy (gliomata Grade III and IV) but of limited extent were to be treated by medium size fields at 250 kv. or 400 kv., since the availability of supervoltage equipment was limited. Fields usually chosen for this technique were two opposing lateral portals measuring 10×8 cm. and a superior portal, 10×6 cm. Figure 1 shows the isodose distribution for this technique.Tumor doses in the range of 5,500 to 6,000 r were to be given in six to seven weeks. Large

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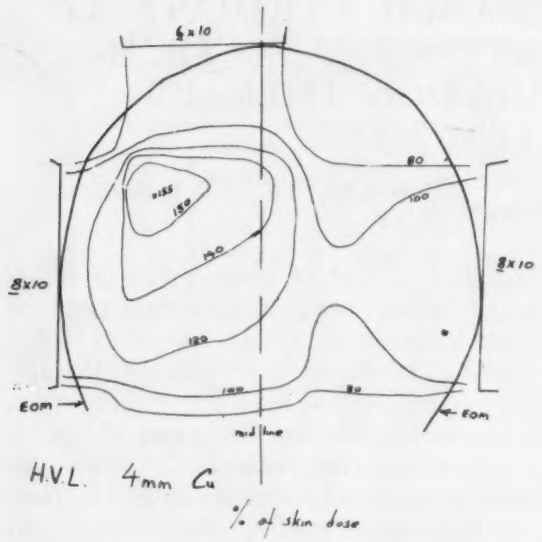

FIG. 1. Isodose distribution (per cent of skin dose) obtained with 400 kv. roentgen-ray apparatus, 4.0 mm. Cu half value layer for portal sizes shown.

field therapy was to be used only in those patients who had evidence of very extensive tumors, regardless of histologic grading. The dose given with the large field technique was to be about 5,000 r in six weeks.

In practice the plan of radiation treatment was adhered to fairly closely, except when the poor condition of the patient precluded the use of accurate small field or rotational techniques. The majority of patients were treated by medium field techniques, a lesser number by rotational small field technique and only a few patients were treated by large field irradiation.

These techniques and doses were chosen because the maximum dose of radiation tolerated by tissue is in inverse relationship to the volume of tissue irradiated. Past experience suggested that gliomata were not particularly radioresponsive. Rather than irradiate large volumes of tissue to a necessarily lower dose, we chose to give higher doses to medium or even quite small volumes of brain tissue if the clinical assessment indicated that the tumor might be encompassed in this volume. It is the purpose of this paper to examine the validity of this concept. An account of the controlled investigation as a whole is to be published later by Penman *et al.*

To this end we have studied the extent of supratentorial gliomata in 30 of the patients in this series who died shortly after their admission to hospital for radiation therapy. The complete clinical notes, roentgenograms and the preserved brains of these patients were available for study. Localization of the tumors and estimation of their size for the purpose of radiation therapy were based primarily on contrast roentgenologic studies. The size and localization of the tumors as indicated by these studies were compared with the actual size and location as shown at postmortem examination. Bull and Rovit[1] have described a classification of the results of localization in roentgenologic diagnosis of brain tumors, dividing them into excellent, good, poor and failed. This classification while satisfactory to the surgeon is of less value to the radiation therapist, since it is probable that a part of the tumor will be underdosed or missed completely if more than a small portion of the tumor is outside the area delineated by the radiologist. Radiation therapy must therefore fail on this account alone. We have modified Bull and Rovit's classification of the results of roentgenologic localization of brain tumors to more adequately suit the purpose of the radiation therapist (Table I). The results of the comparative study of roentgenologic and postmortem findings in the 29 patients who had contrast studies are shown in Table II.

TABLE I

CLASSIFICATION OF RESULTS IN ROENTGENOLOGIC DIAGNOSIS OF BRAIN TUMORS

(1) Excellent—the tumor accurately and completely localized
(2) Good—major portion of the tumor localized, less than 20 per cent of tumor tissue outside, but closely adjacent to the area delineated by the radiologist
(3) Poor—more than 20 per cent of the tumor outside the indicated volume, tumor distantly removed from the area indicated, study inadequate to demonstrate tumor or localize tumor

Table II

ROENTGENOLOGIC DIAGNOSTIC ACCURACY

Localization of Tumor	No. of Cases	Per Cent
Excellent	4	13.8
Good	11	37.9
Poor	14	48.3

The accuracy of roentgenologic localization in this series compares unfavorably with that reported by Bull and Rovit. They reported 50 per cent excellent and 35 per cent good diagnostic accuracy, when the localizing procedures were done within two months of death. Their series, however, only included patients who had had adequate roentgenologic investigations. Many of the patients in this series had had studies which were inadequate, but which were sufficient to indicate the presence of a brain tumor. Following surgery these patients were referred for radiation therapy without further contrast studies. Table III shows the contrast studies performed. The time interval between contrast study and death is shown in Figure 2.

The treatment plans of 21 patients who had received some radiation therapy prior to death were examined. The smallest volume irradiated was a 6.6 cm. sphere. The largest volume was that irradiated by the use of two lateral opposing 15×8 cm. fields and a superior 15×6 cm. field. The portals were selected in each case in the belief that they would encompass the tumor. Failure to include the whole tumor in the irradiated volume could be due to either inadequate portal size or to faulty roentgenologic localization. To investigate our choice of portal size and to exclude failure due to faulty roentgenologic localization, we assumed that the precise center of the tumor had been known. Two additional factors were considered in this analysis: (1) It would be almost impossible to direct the central ray of the treatment portals through the precise center of a tumor even with perfect localization, and an inaccuracy of 1 cm. on either side could occur; and (2)

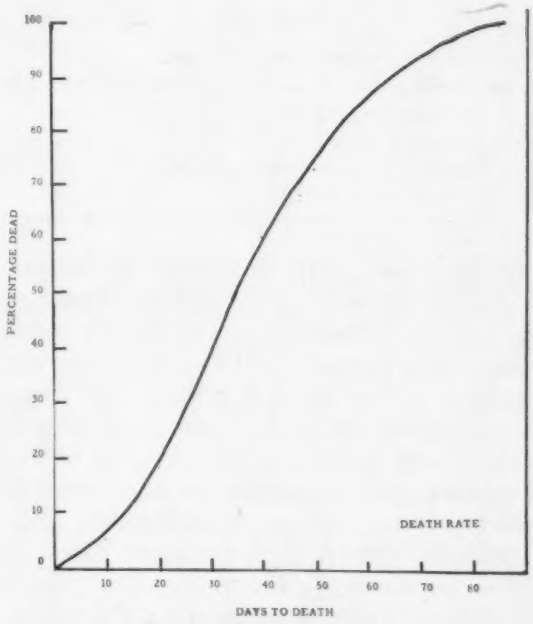

Fig. 2. Time interval between contrast study and death of patients.

a zone of 1 cm. in all directions beyond the apparent extent of the tumor had to be included in the irradiated volume to provide a safety margin of what was thought to be normal tissue. Tumors were considered adequately covered by the treatment plan if these conditions were fulfilled. Tumor coverage was considered questionable if there was the possibility that a portion of the tumor or its surrounding margin of normal tissue was outside the irradiated volume. The tumor was considered missed if a portion of the tumor or of the 1 cm. margin was definitely not within the treated zone. No consideration was given

Table III

ROENTGENOLOGIC CONTRAST STUDIES

Study	No. of Patients
Ventriculography Only	17
Arteriography Only	3
Cystography Only	3
Ventriculography and Carotid Arteriography	4
Cystography and Carotid Arteriography	2

TABLE IV

ACTUAL PORTALS USED

Patients Treated	21
Tumors Covered	2
Tumor Coverage Questionable	11
Tumors Missed	8

in this investigation to possible diffuse microscopic infiltration beyond the localized tumor mass. Isodose curves for the fields used were plotted in the anteroposterior, vertical and transverse planes. The gross tumor extent in each dimension as determined at postmortem examination of the brain was compared with the isodose curve for the corresponding plane. Figure 5 illustrates the results of the analysis for each plane separately, and for the three planes combined. Table IV summarizes the results shown in Figure 5.

We recognize the faults of delineation of cerebral gliomata by gross appearances since microscopic infiltration of tumor cells into adjacent tissue is common. However, even a study such as ours of the gross pathology confirms the well known fact that gliomata spread beyond the confines of certain anatomic areas such as the lobes of the brain and that their exact limits are most difficult to determine. The tumors were almost invariably larger and more extensive than was suspected by the clinical and roentgenologic examinations. The laterally placed tumors showed a spread mainly in an anteroposterior direction, but occasionally extended to the corpus callosum. Extensive spread across the midline was uncommon for the tumors originating laterally. Tumors apparently originating in midline structures, such as the corpus callosum, had rather extensive bilateral involvement. It was also noted that the midline tumors tended to invade the midbrain and pons. Figure 3 is a diagrammatic representation of the tumor in patient No. 18 who had such invasion. Figure 4 is a photograph showing the tumor and its extension into the pons. Two patients in this series had an unsuspected second glioma, widely separated from the tumor the patient was known to have. The double tumors were present in the same hemisphere in one patient and bilaterally in the other. An additional 9 patients had extension of their tumor across the midline.

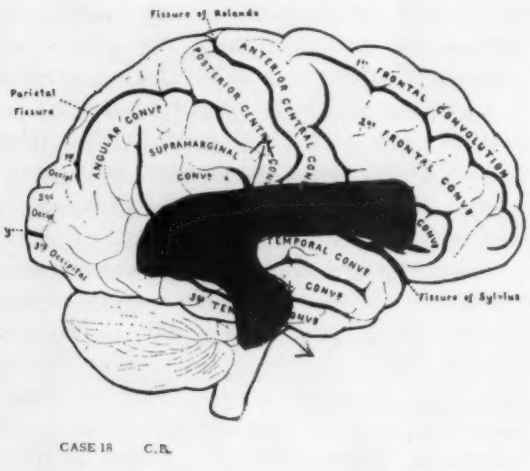

FIG. 3. Diagrammatic representation of extension of tumor with the pons.

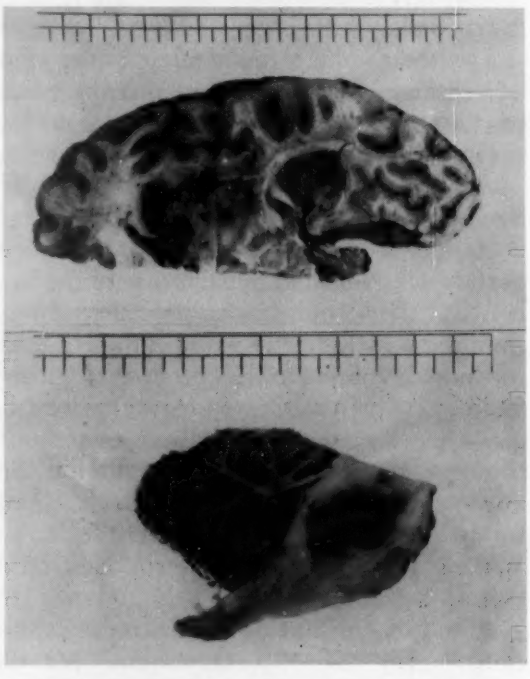

FIG. 4. Photograph of brain sections showing primary tumor and extension with the pons. (Scale in cm.)

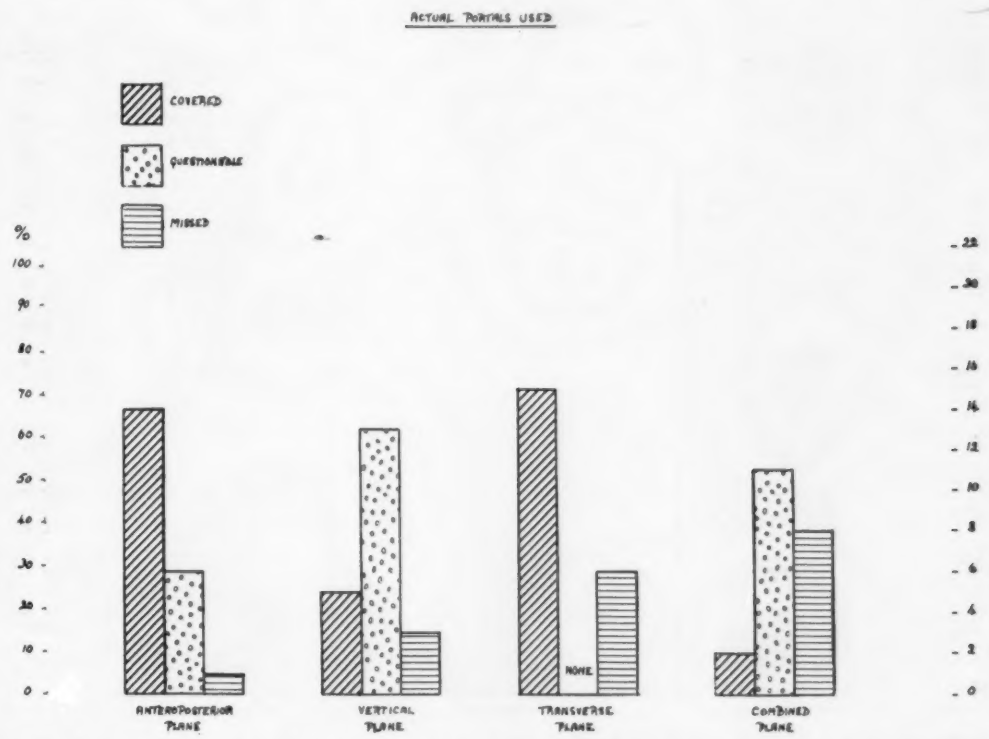

FIG. 5. Graphic representation of the number and percentage of tumors adequately included, questionably included and missed in the volume irradiated. Each plane considered separately and then for all planes combined.

Since most of the patients were treated with medium sized fields, we made a study of the tumor coverage which would have been obtained in the 30 patients with a medium field technique and compared this with the results which would have been achieved by a large field technique. For the purpose of this study, medium fields were defined as two lateral 10×8 cm. portals and a superior 10×6 cm. portal. Large fields were defined as two lateral 12×9 cm. fields and a superior 12×8 cm. portal. The same conditions were applied to this study as in the analysis of the portals actually used. Figures 6, 7 and 8 show the results of a comparison of the degree to which the tumors were included in the irradiated volume for the medium and large field techniques, each plane being considered independently. Figure 9 gives a graphic comparison when all planes are considered together. It clearly shows that had the larger fields been used the tumor would have been adequately covered in nearly three quarters of the patients treated instead of in less than a quarter when the smaller portals were used. The number of patients in whom the tumors would have been missed was reduced from more than one-third to one-fifth. The number of tumors in the questionable group was likewise reduced from one-third to one-tenth. It is apparent from a study of these graphs that this improvement is based mainly on the enlargement of the treatment field in the vertical plane and in the anteroposterior plane. Enlargement of the transverse plane from a 6 to an 8 cm. portal improved the results only slightly.

It must be stressed that this is a highly selected group of patients since all of them died soon after admission. Twenty-seven of the 30 patients had extremely malignant tumors, Grades III or IV. These patients

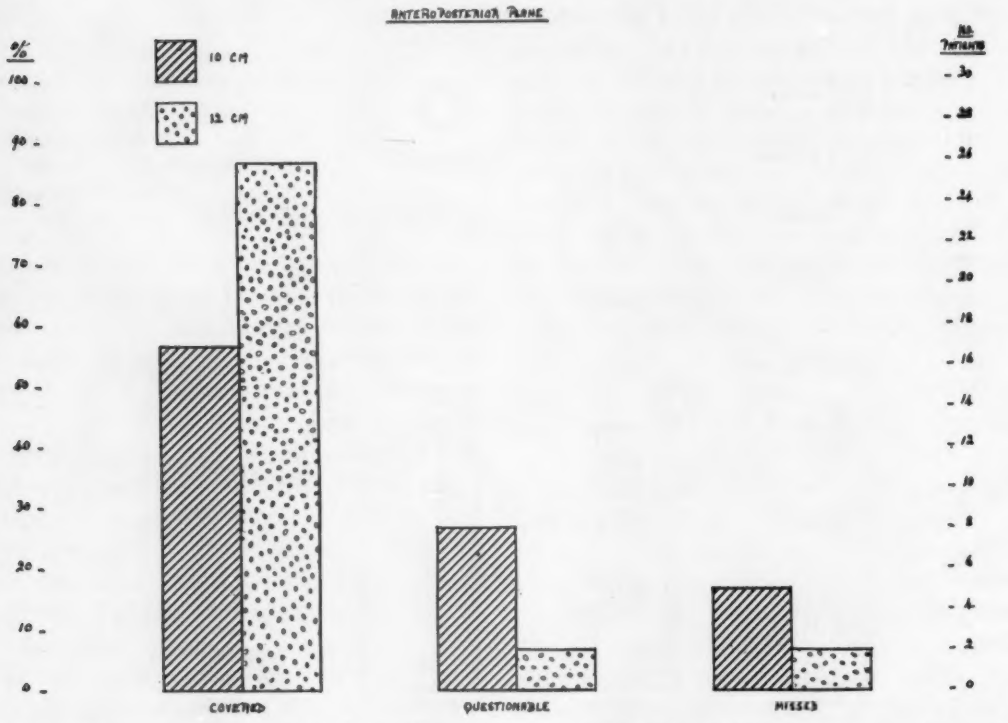

FIG. 6. Graphic comparison of results for medium field versus large field technique for anteroposterior plane.

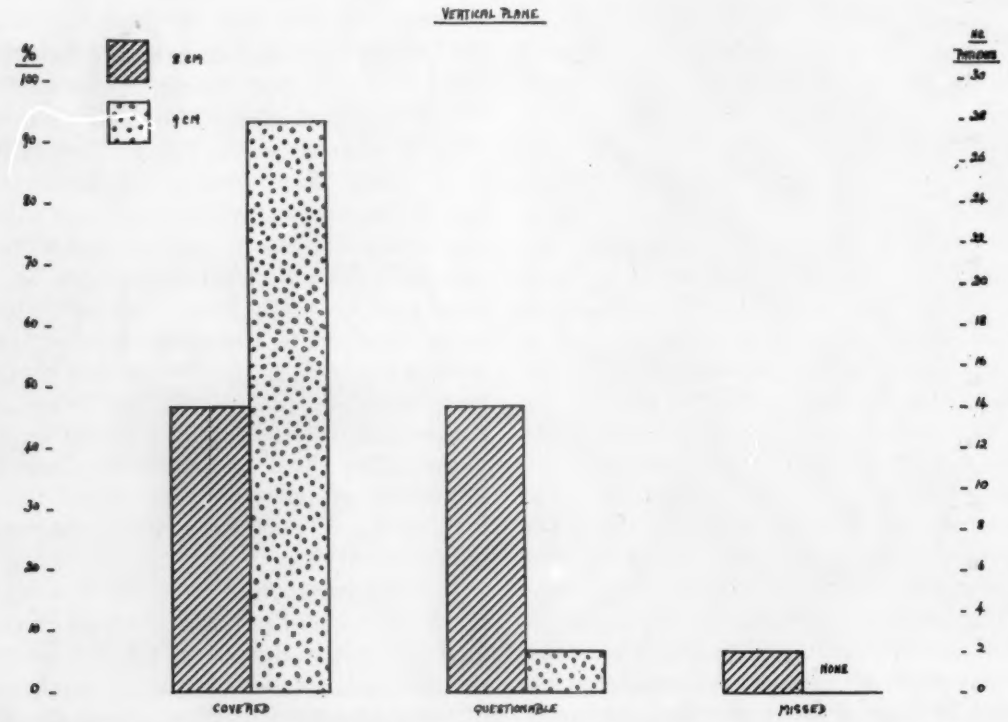

FIG. 7. Graphic comparison of results for medium field versus large field technique for vertical plane.

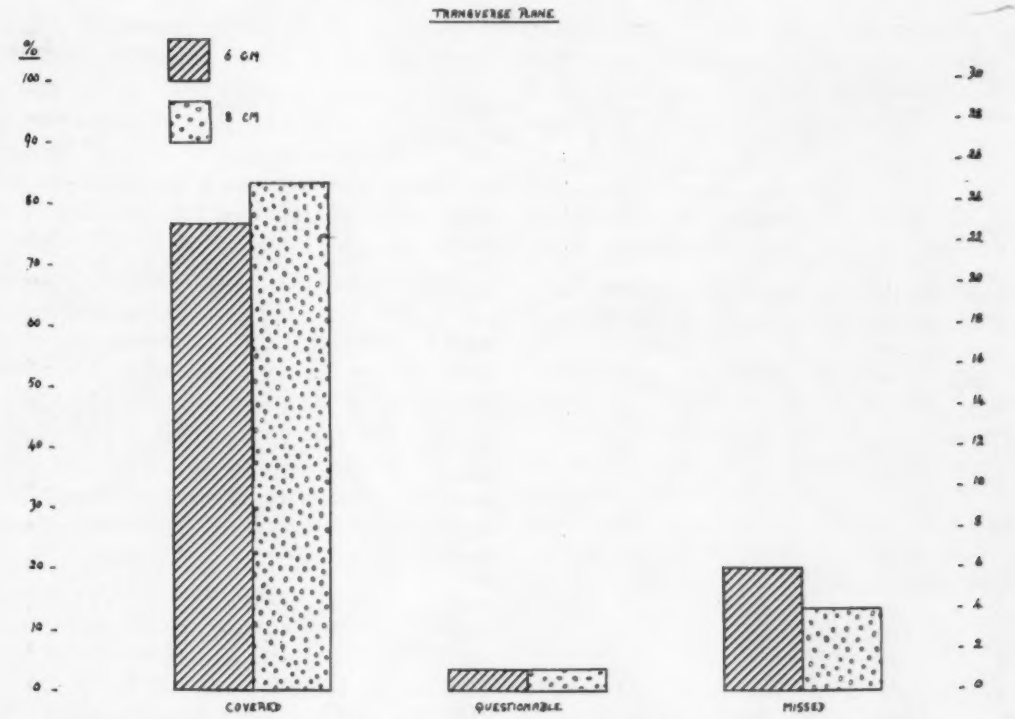

FIG. 8. Graphic comparison of results for medium field versus large field technique for transverse plane.

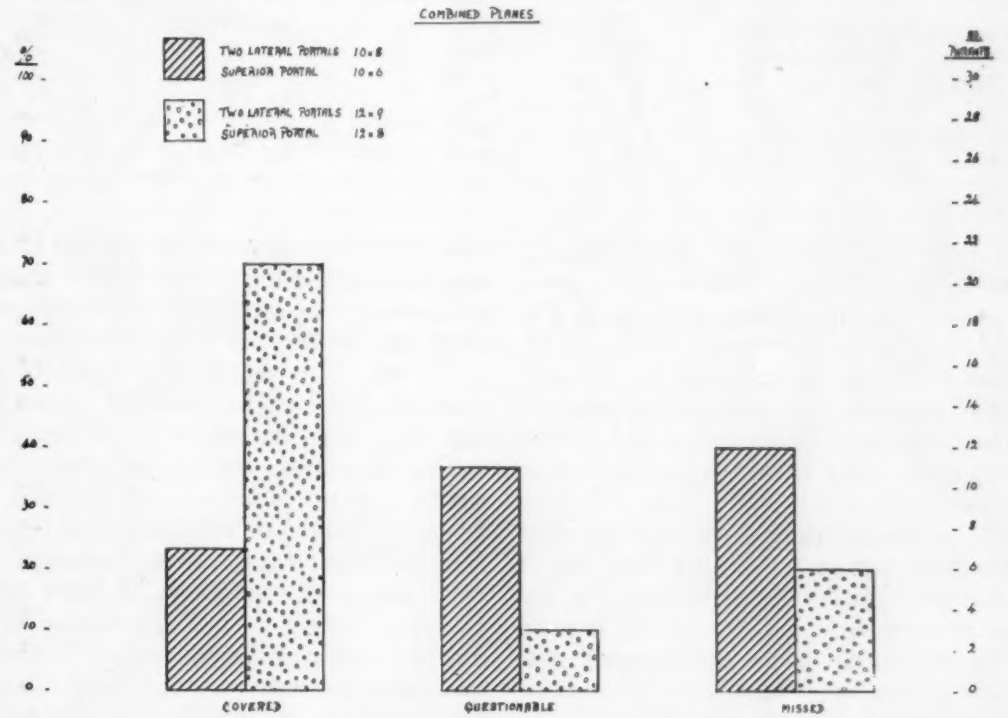

FIG. 9. Graphic comparison of results for medium field versus large field technique for all planes combined.

TABLE V

Case No.	Duration of Symptoms to Surgery	Time of Contrast Study to Death	Histologic Grading	Roentgenologic Localization	Size of Tumor Post Mortem	Size of Tumor plus 1 cm. Margin
1	33 da.	No films	IV	none	7 ×7 × 7.5	9 ×9 × 9.5
2	120 da.	13 da.	IV	good	7.5×3.5× 7.5	9.5×5.5× 9.5
3	180 da.	17 da.	IV	poor	10 ×5 × 4.5	12 ×7 × 6.5
4	210 da.	23 da.	III–IV	excellent	3.5×3.5× 3.5	5.5×5.5× 5.5
5	150 da.	26 da.	III–IV	poor	6 ×4.5× 4.5	8 ×6.5× 6.5
6	37 da.	45 da.	III–IV	failed	3.5×4 × 5.5	5.5×6 × 7.5
7	120 da.	54 da.	IV	good	7 ×5 × 4.5	9 ×7 × 6.5
8	270 da.	38 da.	IV	good	7.5×5 × 5	9.5×7 × 7
9	90 da.	19 da.	IV	poor	7.5×4.5× 4.5	9.5×6.5× 6.5
10	18 da.	33 da.	IV	excellent	6.5×5.5× 7	8.5×7.5× 9
11	90 da.	21 da.	IV	good	6 ×6 × 6	8 ×8 × 8
12	4 da.	37 da.	II	poor	10 ×5 × 5	12 ×7 × 7.5
13	3 yr.	35 da.	III	poor	7.5×4.5× 4.5	9.5×6.5× 6.5
14	120 da.	66 da.	IV	good	7 ×4 × 7	9 ×6 × 9
15	180 da.	45 da.	III–IV	good	7 ×7 × 7	9 ×9 × 9
16	180 da.	22 da.	IV	poor	8.5×5.5× 5	10.5×7.5× 7
17	28 da.	7 da.	III–IV	poor	6.5×4 × 5	8.5×6 × 7
18	28 da.	22 da.	III–IV	poor	9 ×5 × 3.5	11 ×7 × 5.5
19	?	?	IV	poor	13 ×6 × 9	15 ×8 ×11
20	60 da.	52 da.	III–IV	excellent	7 ×3.5× 6	9 ×5.5× 8
21	90 da.	49 da.	II	excellent	4 ×4 × 3.5	6 ×6 × 5.5
22	150 da.	9 da.	IV	poor	12 ×5 × 5	14 ×7 × 7
23	150 da.	48 da.	IV	good	7 ×3.5× 6	9 ×5.5× 8
24	30 da.	17 da.	IV	good	7 ×4 × 4	9 ×6 × 6
25	30 da.	60 da.	IV	poor	7 ×5 × 5	9 ×7 × 7
26	2 yr.	50 da.	III–IV	good	3 ×4 × 2.5	5 ×6 × 4.5
27	180 da.	78 da.	IV	good	5 ×5 × 4.5	7 ×7 × 6.5
28	30 da.	55 da.	III–IV	poor	5 ×3.5× 4	7 ×5.5× 6
29	90 da.	25 da.	IV	good	7 ×5 × 6	9 ×7 × 8
30	4 yr.(?)	66 da.	II	poor	7 ×5 ×10	9 ×7 ×12

may have had larger and more rapidly growing tumors than those who survived for longer periods of time. It is quite unlikely that the tumor size was decreased by radiation therapy in most of the patients treated, since only 6 completed the planned course of therapy. Table v gives the dimension of each tumor as well as other information pertinent to this study.

We conclude that with our present methods of localization and knowledge of spread of gliomata there is no place for small or medium volume radiation therapy in the treatment of the more highly malignant gliomata. If there is to be any hope of success with roentgen therapy, the minimum volume to be adequately irradiated should be at least that volume encompassed by two paired opposing fields measuring 12×9 cm. and a superior portal of 12×6 cm. This means that most of one cerebral hemisphere, midline structures and the medial portion of the other hemisphere should be treated even when the tumor appears to be unilateral. When the lesion is thought to be bilateral or to arise from midline structures, it is necessary to treat the major portions of both hemispheres. If there is clinical evidence to indicate invasion of the cerebral peduncles by neoplasm, it is necessary to include the midbrain and part of the brainstem to cover the

probable extension of the tumor into these structures. Whether roentgen therapy can be effective for these tumors with large volume irradiation is another question; the one answered here is that it is unlikely to be effective if smaller volumes are treated.

We can draw no conclusions about the volume to be irradiated in patients with gliomata Grades I and II, and a similar study for these would be most valuable. When one considers the difficulty of accurate localization and precise centering of the treatment portals, the need to include a margin around the tumor, and the diffuse nature of the neoplastic change in some cases, it is likely that small volume irradiation may even be inadequate for most of the more differentiated and apparently small gliomata Grades I and II.

The point we wish to make is that judgments on the value of irradiation for any group of tumors should include an assessment of the proportion of patients in whom treatment adequate to cover the tumor was given and not rely solely on a statement regarding the dose delivered to some selected point in the volume irradiated.

SUMMARY

Thirty patients with brain tumors referred to the Royal Marsden Hospital for radiation therapy died shortly after admission to the hospital. Postmortem examination of the brains of these patients is the basis of this study. The investigations described indicate that large field techniques are necessary in the treatment of astrocytic gliomata Grades III and IV. Radiation therapy for brain tumors with medium or small field techniques may fail solely through inadequate coverage of the tumor.

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We wish to record our thanks to Professor Smithers in whose department this investigation was done and for his valuable advice and criticism; to Mr. Wylie McKissock and Mr. Logue, who performed the surgery; and to Professor Blackwood, Professor Crawford and Dr. McMenemey, who provided the pathologic diagnoses. An abbreviated report of the work was read at the Seventh International Cancer Congress and a summary is to be published in the Acta of the International Union against Cancer.

REFERENCES

1. Bull, J. W. D., and Rovit, R. L. Radiographic localization of intracerebral gliomata. *J. Fac. Radiologists*, 1957, *8*, 147–157.
2. McWhirter, R. Radiation treatment of cerebral tumours. *Proc. Roy. Soc. Med.*, 1946, *39*, 673–676.
3. Peirce, C. B., and Bouchard, J. Role of radiation therapy in control of malignant neoplasms of brain and brain stem. *Radiology*, 1950, *55*, 337–343.
4. Penman, J. Planning clinical trial of radiotherapy for intracranial gliomata. *Proc. Roy. Soc. Med.*, 1954, *47*, 677–680.
5. Penman, J., and Smith, M. Intracranial Gliomata. Medical Research Council Special Report Series No. 284, Her Majesty's Stationery Office, 1954.
6. Richmond, D. J. Cerebral tumours. *J. Fac. Radiologists*, 1949, *1*, 23–27.

AN EXPERIMENTAL STUDY IN THE USE OF A RAPIDLY DECAYING BETA SOURCE (Pd^{109}) IN THE PRODUCTION OF DEEP CEREBRAL LESIONS*

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IN 1923, Edwards and Bagg[3] produced localized necrotic lesions in the cerebral substance of experimental animals by the use of the naturally radioactive substance, radium and in 1950, Kisieleski *et al.*[7] employed the artificially radioactive isotopes for this purpose.

Pd^{109}, P^{32}, Sr^{90} and Y^{90}, which are almost pure sources of beta emission, have all been studied in laboratory animals[1,2,4,5] and Y^{90} has been used to destroy the pituitary gland in the treatment of mammary carcinoma.[6] Though Pd^{109} has a lower energy of beta emission than the others and, therefore, produces a slightly smaller lesion, it has distinct advantages in that its very rapid rate of disintegration (half life 13.5 hours) eliminates many of the problems of disposal that attend the long active sources. In addition, the convenient form in which it is available (malleable wire) makes it easier to handle than yttrium (ceramic pellets) or phosphorus (incorporated in glass beads).

The present study is of two main parts. These consist of serial observations of the effects produced by Pd^{109} sources of moderate intensity (5 mc), permanently implanted in the brains of cats for periods ranging from one day up to two years, while in the second part Pd^{109} sources of higher intensity (20 mc) were applied for periods ranging from 15 minutes up to 24 hours.

PHYSICAL FACTORS

Pd^{109} is formed in a nuclear reactor from Pd^{108} by capture of a neutron giving a specific activity of about 10 mc per milligram for a 24 hour activation period at a neutron flux of 10^{13} n/sec./cm.^{2}. The decay of this isotope is by emission of beta particles with a maximum energy of 1.0 mev. and a range of about 4 mm. in tissue. This is accompanied by a low energy gamma ray (0.088 mev.) which is 90 per cent converted to electrons, so that for practical purposes, handling and dosimetry, it may be considered a pure beta source. (The Ag^{111} produced from Pd^{110} simultaneously in the Pd target constitutes only 1 per cent of the total activity for a 24 hour activation.)

While the range of beta radiation in tissue is about 4 mm., the distance at which a necrotic lesion may be produced is considerably less. The absorption of Pd^{109} beta radiation in tissue is roughly exponential with a half value layer of about 0.5 mm. and superimposed upon this we must consider also inverse square attenuation.

Because of the unknown amount of self absorption in the source, actual measurements of the radiation field around a small source embedded in lucite were carried out by the photographic method of Tochilin and Golden.[8] The source measured was 1 mm. in length and 0.3 mm. in diameter. At distances greater than 1.0 mm. from the center the isodose surfaces were virtually spherical. When a large source is used (*e.g.*, 3, 4 or 6 mm. of wire), the surrounding field may be estimated by regarding the large source as a series of these small sources. The resultant curves become elongated and are slightly wider in the middle where the individual 1 mm. curves have become superimposed. Very approximately a point 1 mm. from a 20 mc source will receive half a million rads after 90 minutes irradiation, at 2 mm. it will receive 50,000

* From the Division of Neurological Surgery of The University of Chicago and the Argonne Cancer Research Hospital.† Aided by a grant from the Douglas Smith Foundation.
† Operated by the University of Chicago for the United States Atomic Energy Commission.

rads, at 3 mm. it will receive 6,000 rads and at 4 mm. the figure is in the neighborhood of 1,000 rads. It is usually held that an immediate dose of less then 2,000 rads will not produce histologic evidence of permanent injury in brain tissue.

EXPERIMENTS

GROUP 1. PERMANENT Pd^{109} (5 MC) IMPLANTS; 1 DAY TO 2 YEARS

Sixteen cats were used. The isotope was implanted by means of a hollow needle through a small trephine opening (general anesthesia). The animals were sacrificed at gradually increasing intervals (1, 2, 3, 4, 5, 6, 7 and 10 days; 2, 3, and 4 weeks; 3, 6, 12, 18 and 24 months). The brains were perfused with formalin and histologic sections were stained with hematoxylin and phloxine. On the first day there was an area of marked "edema" of approximately 4 mm. diameter with a rather clear line of demarcation between this and normal brain. The intercellular substance was widely vacuolated and stained less deeply than normal. In the center the tissue was fragmented and cell detail was destroyed but the majority of cells elsewhere had preserved their structure. By the third day most of the cells within this area were obviously dead and there began to appear at the periphery a narrow margin of about 0.5 mm. thickness in which the fate of individual cells was uncertain. In this zone the blood brain barrier was broken down as evidenced by permeation of Evans blue dye. About this time a mild edema could be seen spreading around the lesion. It eventually became fairly generalized throughout the hemisphere and was most marked by the fifth and seventh days. By the tenth day it had disappeared. It affected white matter considerably more than it affected gray matter, but its mild character and lack of discrete boundary distinguished it from the disruptive "edema" within the lesion. The absence of a vigorous polymorphonuclear and lymphocytic response was a noticeable feature. By the seventh day macrophages had commenced the task of phagocytosis. Adjacent to the lesion the boundary zone contained dilated capillaries and at this stage of generalized edema it was at its widest (0.5–1.0 mm.). By 2 months phagocytosis was still active but was nearing completion and a cyst had largely replaced the infarct. It had no endothelial lining and its wall was composed of a layer of astrocytes. This was 0.1 mm. wide. At 2 years the lesion was difficult to see with the naked eye but was easily identified by the microscope (Fig. 1*D*). It was a cyst 3 mm. long and 1.5 mm. wide in the fixed state. Its capsule was a thin layer of astrocytes outside which the cerebral architecture appeared perfectly normal.

GROUP 2. TEMPORARY Pd^{109} (20 MC) IMPLANTS; 15 MINUTES TO 24 HOURS

Four dogs and 4 cats were used.

Animal 1. Eight sources were placed in No. PE 50 polyethylene tubes and inserted in the animal's brain (generalized anesthesia) for 3, 6, 9, 12, 15, 18, 21 and 24 hour periods and then removed. The animal was sacrificed 8 days later. All the lesions in the formalin hardened state were of 5–6 mm. diameter except one (Fig. 2, top). This source had become displaced during the procedure and was replaced. The repositioning was not exact and it had in effect created a double lesion.

Animal 2. Eight similar sources were placed for 15, 30, 45, 60, 75, 90, 105 and 120 minutes, and the animal was sacrificed 10 days later. The first visible lesion corresponded to the 45 minute implant. The 60 and 75 minute lesions were progressively larger and the 90 minute necrosis was of maximum size (5–6 mm. diameter). There was no further increase with the 105 and 120 minute implants (Fig. 2, bottom).

Animals 3 and 4. These animals had similar lesions and were sacrificed immediately. (It should be noted that the shorter duration implants were inserted last and so all arrive at the end of their period of irradiation at the moment of sacrifice.) The first macroscopic lesion was evident at 105 minutes and the first microscopic lesion at 75 minutes (Fig. 1 *A*). Before that time it

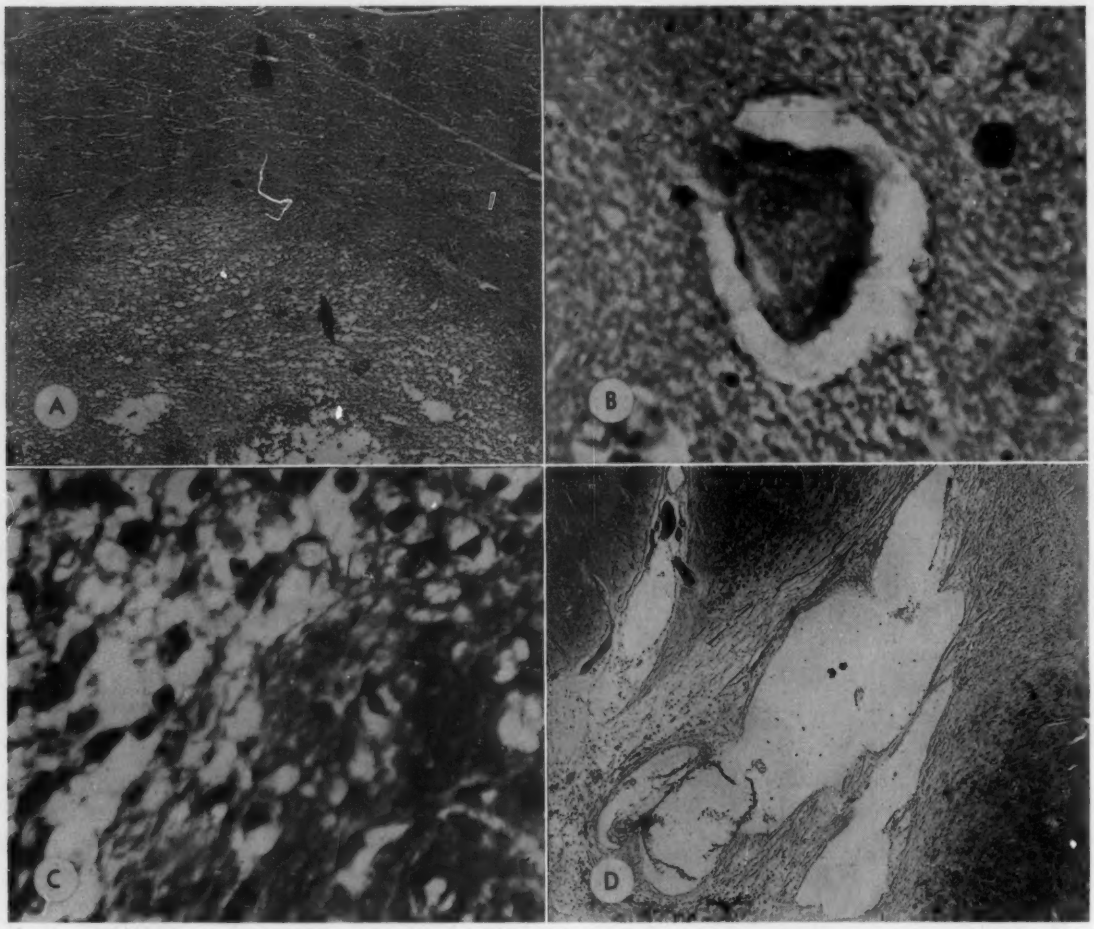

FIG. 1. (*A*) Demonstrates periphery of area of disruptive "edema." (Pd^{109} 20 mc, 105 minutes irradiation. Immediate sacrifice.) (*B*) Cell within the area of "edema" shown in *A*. This cell, situated 2 mm. from the source, has received more than 100,000 rads. It will become completely necrotic. (*C*) Demonstrates abrupt transition from necrosis into boundary zone. The transition between boundary zone and normal tissue is equally abrupt. (Pd^{109} 20 mc, 3 hours. Sacrificed 14 days later.) (*D*) Cyst 2 years after implantation. (Pd^{109} 5 mc. Complete decay.)

was not possible to tell if the edema which was present was due to the irradiation or the trauma of insertion. At that period cell destruction was apparent in the center.

Animals 5, 6, 7 and 8. These animals had 6 instead of 8 lesions and sacrifice was made at $1\frac{1}{2}$, 4, 6 and 14 days afterwards. These confirmed the early development of the lesion and differed from the animals of Group 1 in that there was a vigorous polymorphonuclear and lymphocytic response to the necrosis. In the $1\frac{1}{2}$ day sections blood vessels still seemed to function in areas otherwise rather dead and around these many inflammatory cells could be seen.

GROUP 3. PLACEMENT OF Pd^{109} (20 MC) IN THE REGION OF THE INTERNAL CAPSULE OF MONKEYS TO INVESTIGATE THE POSSIBILITY OF IMMEDIATE OR SUBSEQUENT NEURONAL DAMAGE OUTSIDE THE AREA OF NECROSIS

Three animals were used (general anesthesia). An isotope was stereotactically implanted adjacent to each internal capsule (globus pallidus). No hemiplegia resulted. At a subsequent occasion a further juxtacapsular implant was made on one side and an intracapsular implant on the other. Hemiplegia developed on the side corresponding to the intracapsular placement and was maximum on awakening

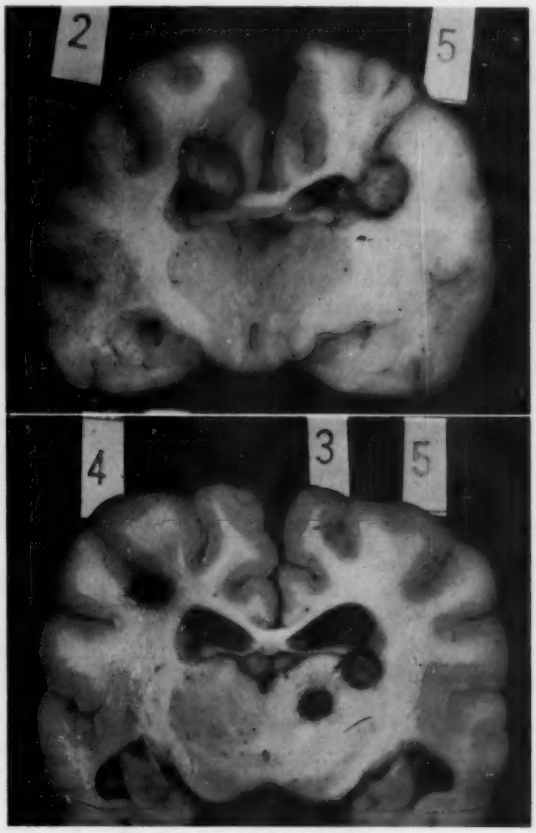

Fig. 2. *Top.* Brain slice of Animal 1, Group 2, Pd^{109} 20 mc. Sacrificed at 8 days. (*2*—6 hours, *5*—15 hours irradiation.) *Bottom.* Brain slice of Animal 2, Group 2, Pd^{109} 20 mc. Sacrificed at 10 days. (*3*—45 minutes, *4*—60 minutes, *5*—75 minutes irradiation.)

from anesthesia (about 3 hours later). Two animals were sacrificed after one week to confirm the location of the isotopes (Fig. 3). A third animal still survives nearly 3 years later. No further paralysis has developed.

GROUP 4.

One final experiment was performed to confirm that the size of the lesion would depend upon the length of wire used as well as upon the duration and intensity of irradiation. Identical wires of 2, 3, 4 and 6 mm. lengths were implanted in a single animal and the size of the lesions was as predicted on physical data (Fig 4).

DISCUSSION

As one might have predicted from the physical data, lesions of maximum size (5–6

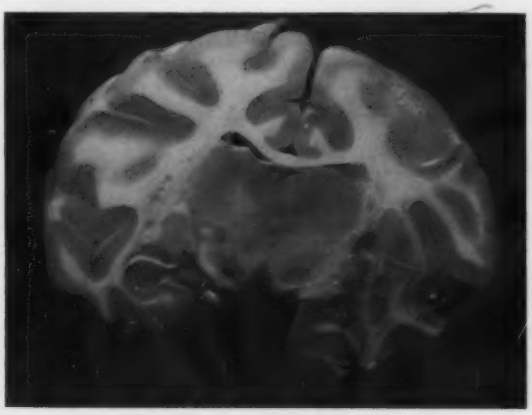

Fig. 3. Brain slice of Animal 2, Group 3, Pd^{109} 20 mc. Sacrificed after 1 week. These two lesions close to the internal capusle did not cause hemiplegia.

mm. in diameter) were produced by the higher energy sources (20 mc) after 90 minutes irradiation. The moment at which functional activity ceased within this tissue is unknown. It is not conceivable that it could continue after the presence of histologically verifiable disruption and it

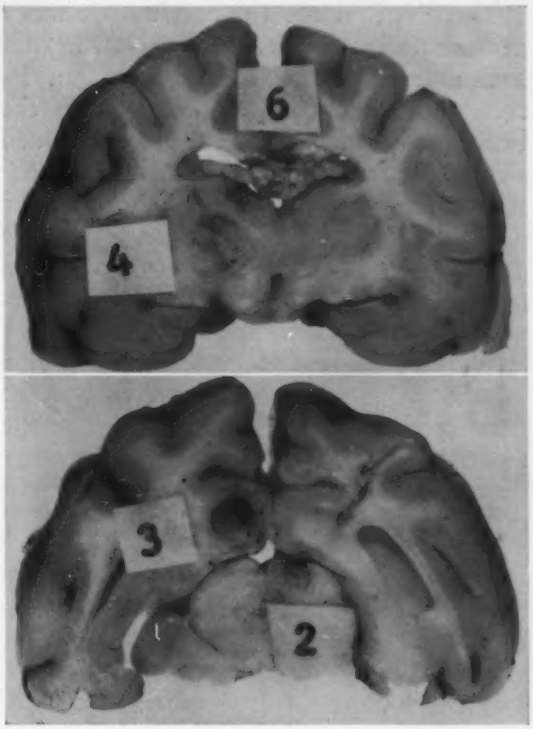

Fig. 4. Brain slice, Cat Experiment 4. Pd^{109}, 3 hours irradiation. Sacrificed at 14 days. Shows relative sizes of lesions produced by pieces of wire 2, 3, 4 and 6 mm. long.

is, therefore, regarded as complete by 90 minutes. Whether cessation of function occurs before, as is likely, or was concomitant with the development of disruption, it is apparent that its occurrence might denote clinically the correctness or incorrectness of placement of the source. If incorrect, the isotope could be withdrawn before the maximum lesion was produced. If withdrawn before 45 minutes no macroscopic lesion would appear.

The calculation of physical data also indicated that no immediate or late injury would occur outside the area of immediate necrosis, and histologic examination of Group 1 animals demonstrated that no further anatomic lesion developed over the 2 year period of study though the 5 mc sources remained until full decay. The absence of early or late sign of neurologic dysfunction (in the animals of Group 3) outside the area of necrosis further confirmed this extremely circumscribed nature of the injury.

These lesions are the result of primary necrosis and are unlike the late injuries that sometimes follow excessive roentgen irradiation therapeutically directed to the brain. They can be made to predetermined size according to the intensity of the source, length of the source (wire) and the duration of irradiation chosen. The slow development of the necrosis provides a measure of safety in cases of misplacement. There is no danger of immediate or late injury beyond the calculated volume of tissue. Where precise destruction of deep cerebral tissues is required as in the treatment of Parkinson's disease, beta-ray necrosis may prove a safe and convenient method.

SUMMARY

1. Necrotic lesions have been created in the deep cerebral substance of laboratory animals by the radioactive isotope Pd^{109}, and the lesions have been studied histologically.

2. The periods of irradiation varied from 15 minutes up to complete decay of the isotope and the survival periods of the animals ranged from immediate sacrifice up to almost 3 years observation.

3. Using a source of high intensity (20 mc) the lesion is first detected after 45 minutes irradiation and reaches its maximum of 5–6 mm. diameter after 90 minutes. There is no evidence of immediate or late extension of neuronal damage outside this area of necrosis.

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REFERENCES

1. BAILEY, O. T., BOYESEN, S., and CAMPBELL, J. B. Beta radiation of feline caudate nucleus: late results. *J. Neurosurg.*, 1957, *14*, 536–542.
2. BOYESEN, S., and CAMPBELL, J. B. Stereotaxic implantation of calibrated Pd^{109} and Y^{90} spheres: technique for producing predictable subcortical lesions in brains of laboratory animals. *Yale J. Biol. & Med.*, 1955–56, *28*, 216–224.
3. EDWARDS, D. J., and BAGG, H. J. Lesions of corpus striatum by radium emanation and accompanying structural and functional changes. *Am. J. Physiol.*, 1923, *65*, 162–173.
4. HARPER, P. V., LATHROP, K. A., and KENNEDY, T. Radiation dosage in tissue surrounding small beta-ray sources. Rare Earths in Biochemical and Medical Research; conference sponsored by the Medical Division Oak Ridge Institute of Nuclear Studies, Oct., 1955. Available from Office of Technical Services, Department of Commerce, Washington 25, D. C.
5. HARPER, P. V., RASMUSSEN, T. B., and KENNEDY, T. Reaction of brain tissue to beta radiation. Rare Earths in Biochemical and Medical Research; conference sponsored by the Medical Division Oak Ridge Institute of Nuclear Studies, Oct., 1955. Available from Office of Technical Services, Department of Commerce, Washington 25, D. C.
6. KELLY, W. A., EVANS, J. P., HARPER, P. V., and HUMPHREYS, E. M. Effect upon hypophysis of radioactive yttrium. *Surg., Gynec. & Obst.*, 1958, *106*, 600–604.
7. KISIELESKI, W., SVIHLE, G., and BRUES, A. M. Preparation of radioactive glass beads. *Science*, 1950, *112*, 400–402.
8. TOCHILIN, E., and GOLDEN, R. Film measurement of beta ray depth dose. *Nucleonics*, 1953, *11*, 26.

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RADIOLOGIC ASPECTS OF CHEMOPALLIDECTOMY AND CHEMOTHALAMECTOMY*

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THE introduction of chemopallidectomy and chemothalamectomy[1] has led to the development of a new roentgenologic technique essential for proper anatomic placement of the surgical lesion. This technique has been used by the authors in approximately 150 surgical procedures, and the clinical assessment of the results of these procedures in 118 of the patients is described elsewhere.[3] Observations available at present indicate that the thalamus is a more favorable site of treatment than is the globus pallidus.

The following description of the roentgenologic technique illustrates the simplicity of the procedure and of the equipment required. It can readily be performed in most radiologic departments.

ROENTGENOLOGIC EXAMINATION

The purpose of the roentgenologic examination is localization of the chemopallidectomy cannula to enable the surgeon to guide it to the target; in brief, localization of the treatment site. A series of roentgenograms in the frontal and lateral projections serves as a three-dimensional orientation. The points of reference are the foramen of Monro, the midline of the patient's skull, and the third ventricle.

The technique used by the authors is similar to that of Cooper,[2] but with changes that have been found to simplify the procedure.

The greater part of the procedure is performed in the operating room with the patient supine under local anesthesia. The positioning of the patient with parkinsonism on the operating table is sometimes a problem because of tremor, rigidity or extreme kyphosis which may be alleviated by supporting him with pillows or blankets. During this preparatory period a wood "tunnel" is also placed under the patient's head to accommodate the cassette for the frontal projection. In addition the head is supported and lifted off the underlying radiopaque headrest by means of a cotton and gauze doughnut.

A sagittal line is drawn on the head from the nasion to the lambda and a coronal line at right angles to it about 13 cm. behind the nasion extending down over the temples just in front of the tragus bilaterally. The sagittal line enables the technician to position the patient accurately in the frontal projection (modified Chamberlain-Towne) despite the surgical drapes. The surgical target behind the foramen of Monro will be close to the plane of the coronal line which is used as an external landmark to position the chemopallidectomy cannula. When the bur hole is made in the coronal line, a needle passed through the hole and aimed in line with the coronal plane will enter the basal ganglion at approximately the desired distance posterior to the foramen of Monro.

Following the placement of the bur hole approximately 6 cm. from the midline in the coronal plane, air is introduced directly into the lateral ventricle through a ventricular cannula inserted through the bur hole. The direct ventricular injection is less uncomfortable for the patient and assures better filling of the ventricular system than the injection into the lumbar subarachnoid space for lumbar pneumoencephalography.

The chemopallidectomy needle is positioned utilizing the external landmarks, *i.e.*, the bur hole, the coronal line, and a point above the eyebrow, approximately

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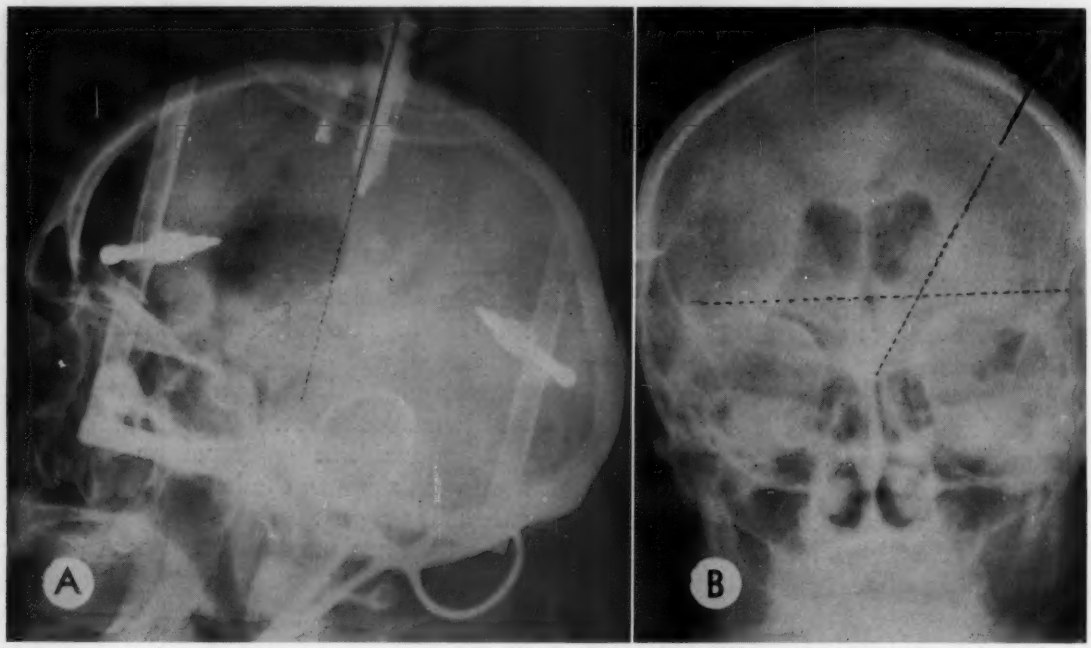

FIG. 1. (*A*) Preliminary placement of cannula and (*B*) projection of axis of needle.

1½ to 2 cm. to the same side of the midline. In order to keep the chemopallidectomy needle in a fixed but changeable position during the procedure, the authors have used the head-fixation device described by Cooper.[2]

The first set of roentgenograms is made after the needle with the metallic stylet has been positioned according to the external landmarks and inserted a few millimeters through the dura. With the patient in the brow-up position, roentgenograms are made in the frontal and lateral projections using a Lysholm grid cassette to improve detail. The usual factors are: 70 to 80 kv., 1/10 to 3/10 sec., at 36 inches distance. The central beam for the lateral projection is about 2 to 3 cm. above a line from the external canthus of the eye on the external auditory canal. The central beam for the frontal projection is just above the nasion. The roentgenograms are processed immediately through rapid solutions and are examined while they are still wet. The metallic stylet is inspected, and by projection of the axis, the eventual position in relation to the foramen of Monro and the midline is estimated (Fig. 1, *A* and *B*). If necessary, the direction of the stylet is changed to produce the desired eventual placement and another set of roentgenograms is taken.

When a line drawn through the needle passes through the target, the cannula is advanced to the proper depth. Another set of roentgenograms is taken to confirm the correct placement of the cannula (Fig. 2, *A* and *B*). Measurements are made on the wet films: (1) in the lateral projection: from the posterior rim of the foramen of Monro to the tip of the stylet; (2) in the frontal projection: from the midline to the tip of the stylet; and finally (3) the depth of the cannula is determined in relation to the third ventricle, either on the frontal or the lateral projection.

The authors have considered a cannula to be in the anterior globus when it is placed from 0 to 6 mm. posterior to the foramen of Monro and 12 to 18 mm. lateral to the midline; in the posterior globus, when it is placed from 7 to 15 mm. behind the foramen of Monro and 17 to 25 mm. lateral to the midline; and in the thalamus, when it is placed within 13 to 18 mm. posterior to the foramen of Monro and the same distance lateral to the midline. The position in the craniocaudad axis is at the

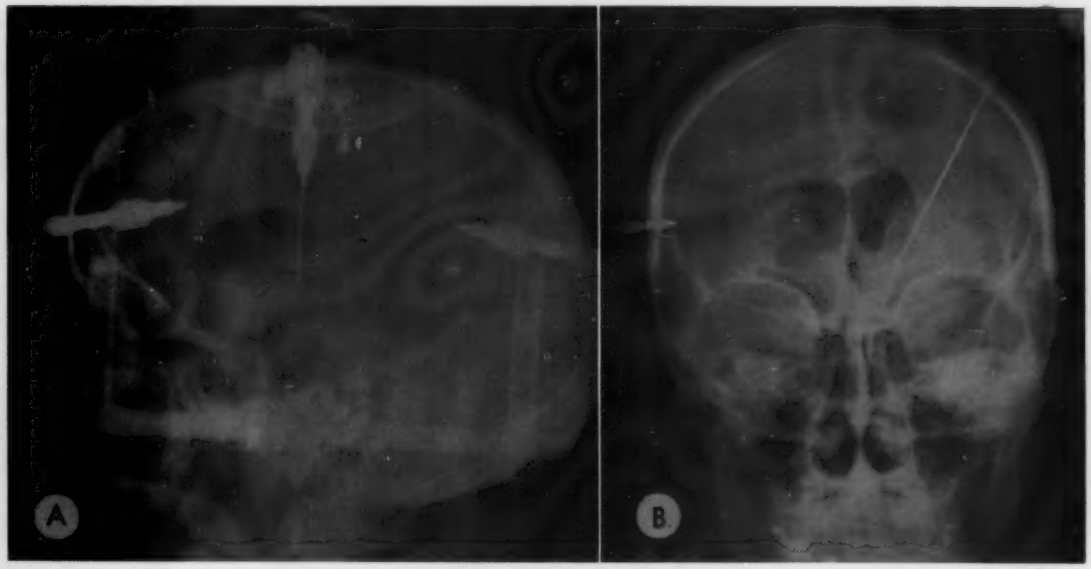

Fig. 2. (*A* and *B*) Final placement of cannula.

level of the middle of the third ventricle or slightly higher. The cannula can usually be introduced into the desired position and its correct placement ascertained by means of three sets of roentgenograms.

Following the completion of the entire procedure, control roentgenograms are made to verify the final position of the treatment site. At this time the alcohol mixed with a small amount of pantopaque has been injected and the metal stylet replaced by a flexible plastic stylet graduated in centimeters. In addition, a tiny fragment of metal wire introduced through the chemopallidectomy needle permanently marks the treatment site.

One or more injections through the indwelling cannula are usually required during the first and second weeks after operation; and before each injection, roentgenograms are made in the frontal and lateral projections to ascertain that the position of the needle has not changed. The pantopaque from the first injection is usually still visible, although there are considerable individual variations in the diffusion of the material.

ROENTGENOLOGIC DEMONSTRATION OF COMPLICATIONS

Among the complications encountered during the treatment of 118 patients reported elsewhere[3] have been intracerebral hematoma and abscess. These complications usually can be diagnosed or at least suspected by means of roentgenograms taken postoperatively while the cannula is still in place in the brain. A hematoma or abscess usually develops in the depths of the brain around the tip of the cannula and causes a shift of the cannula. A comparison of the roentgenograms obtained during surgery with those obtained after surgery readily demonstrates this shift. Therefore, whenever such a complication is suspected because of the clinical condition of the patient, frontal and lateral roentgenograms of the skull are taken and are compared with those obtained during surgery. It is unusual to see any great displacement of the cannula in patients without clinical signs of complications, although a few millimeters of displacement have occasionally been found in such patients and have been thought to be due to a mild localized edema caused by the injection.

An intracerebral hematoma or abscess can sometimes be suspected not only from displacement of the needle but also from curvature of the needle in the brain. A catheter that is not displaced has a straight course in brain tissue as when it was in-

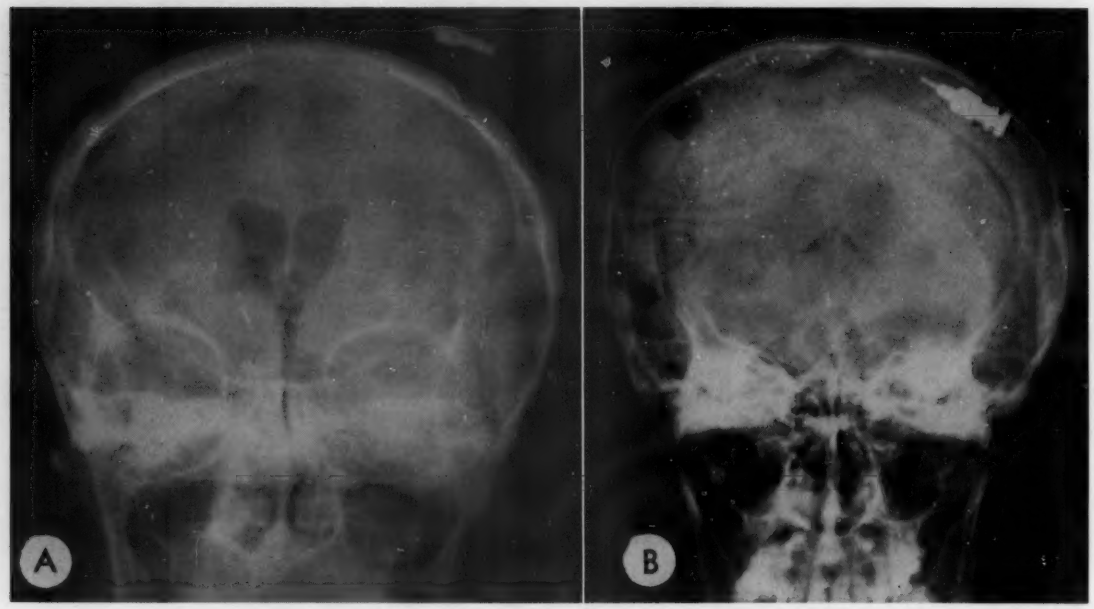

FIG. 3. Case I. Medial displacement of cannula by intracerebral hematoma. (*A*) Placement at operation. (*B*) Position thirteen days postoperatively.

serted at the time of surgery, whereas a catheter that has been displaced will have a somewhat curved course due to the bending of the catheter in the brain. Brief summaries of five cases illustrate these facts.

ILLUSTRATIVE CASES

CASE I. This patient was somewhat somnolent following surgery. Ten days postoperatively, her condition suddenly worsened, and frontal and lateral roentgenograms of the head were taken. The lateral roentgenograms were not remarkable, but a definite displacement of the cannula in a medial direction was seen in the frontal projection (Fig. 3*B*), as compared with the roentgenogram taken at the time of surgery (Fig. 3*A*). The metal marker placed in the treatment site also was displaced and found lying almost exactly in the midline. This displacement was much greater than would be anticipated from brain edema; a craniotomy was therefore performed and a large intracerebral blood clot removed.

CASE II. This patient did well for a few days after surgery, but gradually became more and more comatose and extremely rigid with contralateral hemiparesis. A control roentgenogram (Fig. 4*B*) showed a lateral displacement of the cannula in the frontal projection as compared to the original roentgenogram (Fig. 4*A*). Craniotomy revealed a huge intracerebral hematoma.

CASE III. Another patient, from whom the cannula had been removed, developed hemiparesis. Pneumoencephalography was therefore performed which revealed a displacement of the third ventricle away from the injection site, suggesting a hematoma (Fig. 5, *A* and *B*). However, since there was only little displacement of the lateral ventricles, it was decided not to operate. The patient's condition improved somewhat, although he never made a good recovery.

CASE IV. An unusual complication was found in a fifty-four year old woman, who, immediately after operation, was unable to move the contralateral hand and who, on the eighth postoperative day, had a right-sided convulsion followed by complete paralysis of the right arm. A pneumoencephalogram was obtained at this time but showed only poor filling and no definite shift of the ventricular system. Two weeks later the patient's condition greatly deteriorated over a few days and a complete right hemiparesis developed. Pneumoencephalograms obtained at this time showed a large cyst in the left cerebral hemisphere, communicating with the ventricular system (Fig. 6, *A* and *B*). This cyst was apparently under some tension inasmuch as it has displaced the ventricular system

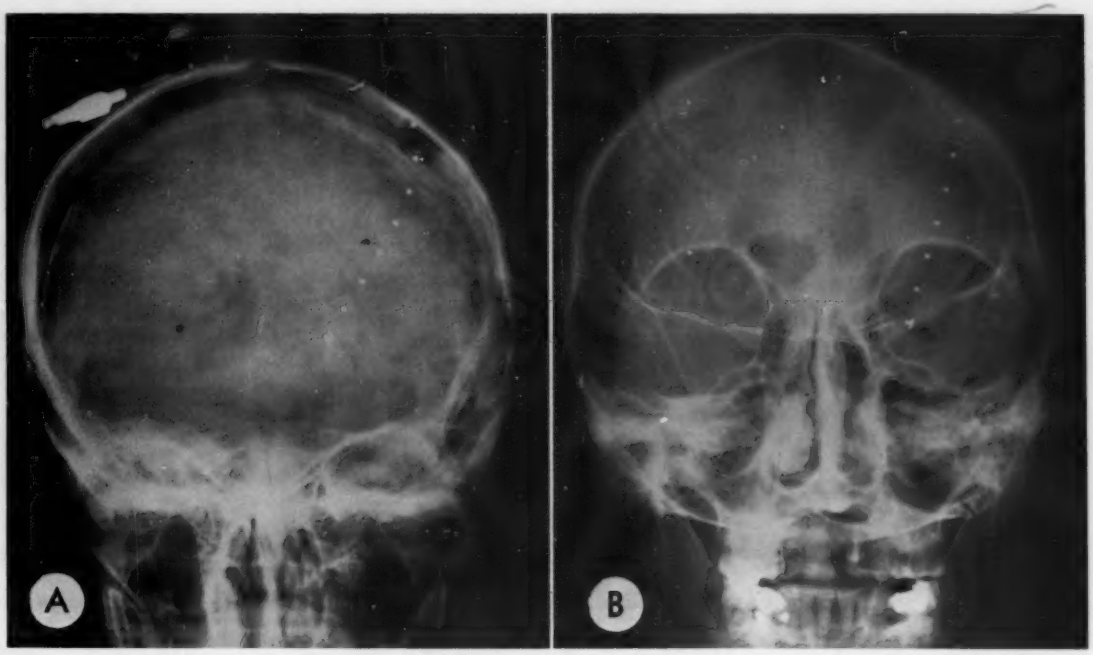

Fig. 4. Case II. Lateral displacement of cannula by hematoma. (*A*) Placement at operation. (*B*) Position two days postoperatively.

toward the opposite side and depressed the ipsilateral lateral ventricle. A craniotomy was done. A huge, air-filled cyst was found which extended from the basal ganglion upward close to the vertex. Although not proved, it is believed that the cyst resulted from necrosis, probably caused by injection of the alcohol into an artery during surgery.

Case V. It is occasionally found that the injected material spreads much more widely than anticipated; and the case of a sixty-two

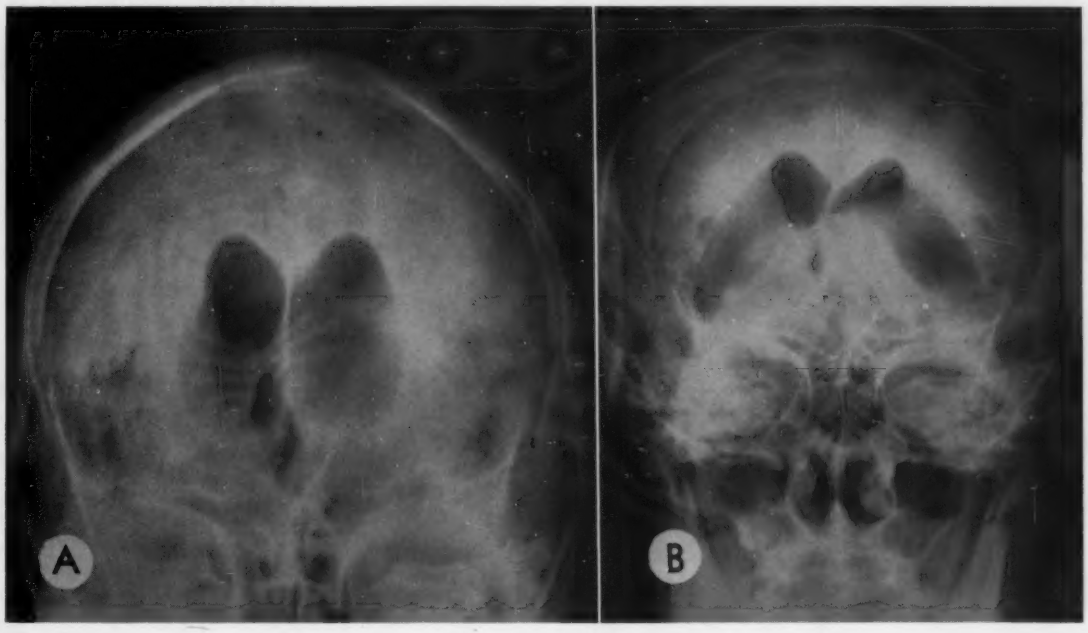

Fig. 5. Case III. (*A* and *B*) Displacement of third ventricle by small hematoma (?).

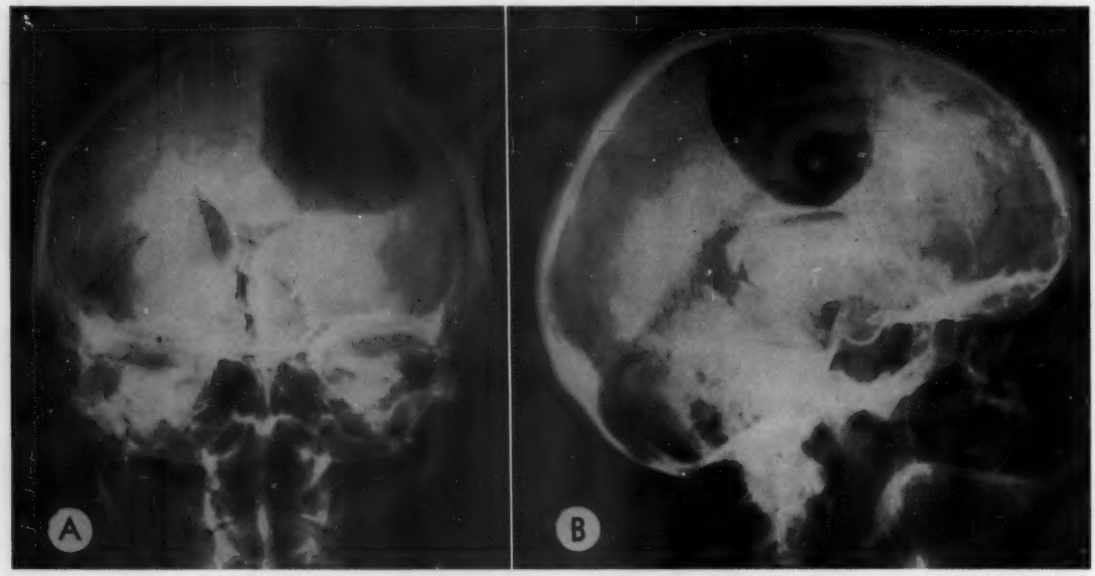

FIG. 6. Case IV. (*A* and *B*) Air-filled cyst in left hemisphere.

year old man demonstrates this fact (Fig. 7). In this case an unusually large amount of alcohol mixed with pantopaque (3 cc.) was injected because of the persistent symptoms, and the roentgenograms showed that the material has spread beyond the midline into the opposite hemisphere. In addition to this complication, the patient developed meningitis and died.

FIG. 7. Case V. Spread of injected material across midline.

In our later series improvement in roentgenologic technique and better demonstration of anatomic landmarks on ventriculograms has resulted in more accurate evaluation of the placement of the cannulas. The relation between the placement of the cannula and the clinical results following surgery has been evaluated elsewhere by the authors.[3]

SUMMARY

The roentgenologic aspects of chemopallidectomy and chemothalamectomy and of certain postoperative complications are described. It is emphasized that an accurate localization of the treatment site can be accomplished by rather simple means.

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REFERENCES

1. COOPER, I. S. Intracerebral injection of procaine into globus pallidus in hyperkinetic disorders. *Science*, 1954, *119*, 417–418.
2. COOPER, I. S. The Neurosurgical Alleviation of Parkinsonism. Charles C Thomas, Publisher, Springfield, Ill., 1956, p. 104.
3. TAARNHØJ, P., ARNOIS, D-C., and DONAHUE, L. A. Chemopallidectomy as treatment for Parkinson's disease; evaluating results in 118 patients. *J. Neurosurg.* In press.

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THE VALUE OF ROUTINE PLAIN ROENTGENOGRAMS IN THE DIAGNOSIS OF SACRAL PERINEURIAL CYSTS*

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PERINEURIAL sacral cysts are not uncommon. Tarlov[13] encountered the condition 5 times in 30 dissections of the sacral nerve roots and ganglia. Two of the examples here shown (Fig. 1, *A*, *B* and *C;* and 2, *A*, *B* and *C*) were found in the study of approximstely 40 cadavers.

Some of these cysts are known to produce sciatic symptoms which may be mistaken for those of radiculitis or of pressure from a herniated lumbosacral disk. Backache, pain, hyperesthesia and loss of muscle power in the thigh as well as absent ankle jerks and deficits of sacral nerve function are to be mentioned. Some patients are tender to deep pressure over the sacrum. There may be a history of trauma. Tarlov[13] states: "Cysts produce syndromes referable to the sciatic nerve or the caudal nerve roots—paresthesias of the penis or vagina, urinary disturbance and sensory changes over the buttocks and perineal area." Either urinary retention or incontinence may be complained of. Symptoms are frequently progressive.

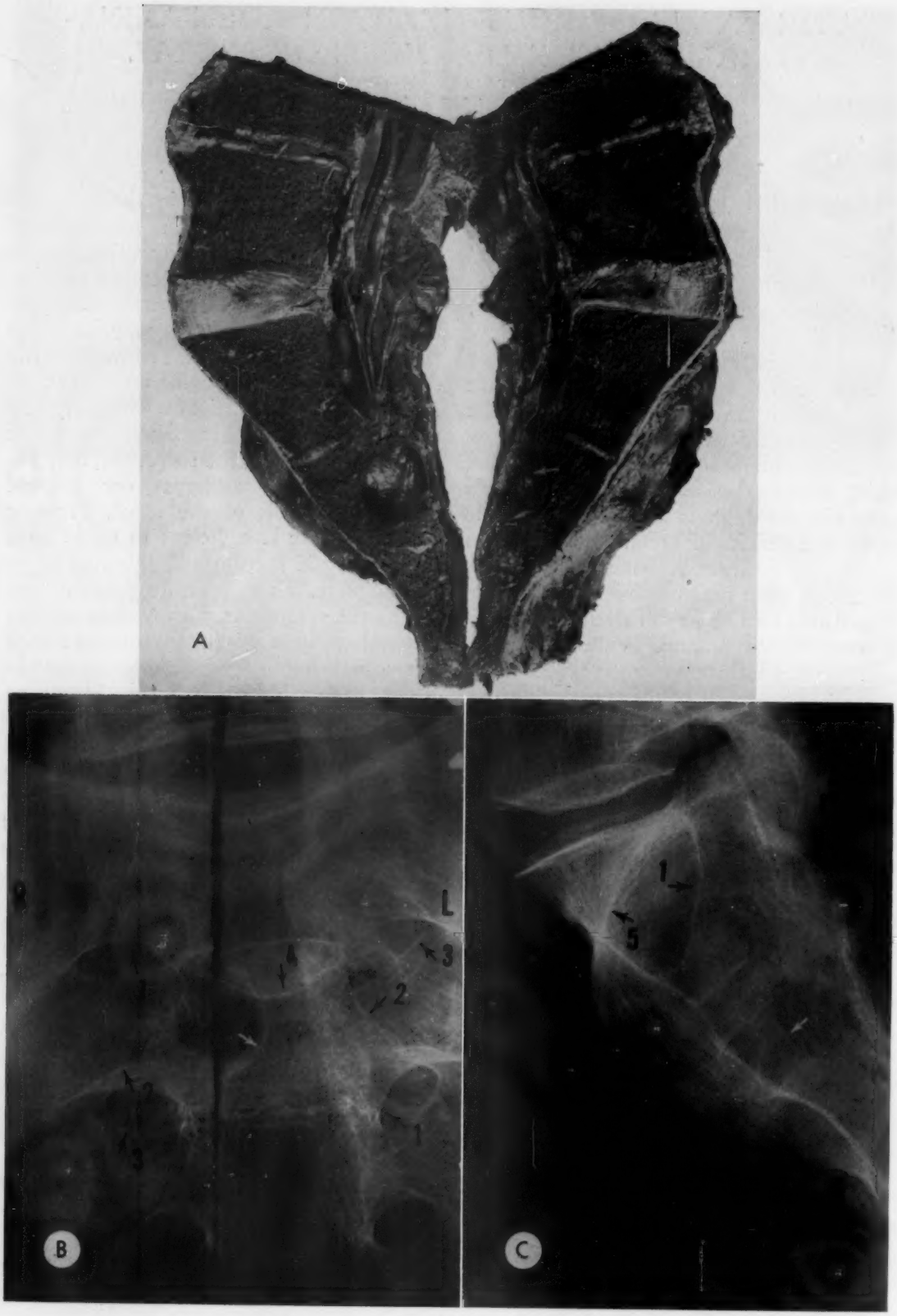
This common condition may be visualized without myelography and should be sought for on plain roentgenograms of the sacrum.

Certain authors[5,8,11] contrast the perineurial cysts with extradural or meningeal cysts which are formed by a herniation of the arachnoid through a defect in the dura. These may be congenital or caused by operative trauma. The perineurial sacral cysts, on the contrary, usually form over the dorsal surface of the posterior root at the junction with the dorsal ganglion and entirely outside of the dura. The ventral root may be uninvolved at first. Tarlov[13] believes that they develop in the potential subperineurial space from subarachnoid hemorrhage or cell degeneration, while Rexed and Wennström[6] describe the formation of small cysts by arachnoidal proliferation. The nerve fibers may be inside the lumen or lie within the wall of the structure. As gradual enlargement occurs, they become stretched over the periphery of the sac. Also, other sacral nerves, the filum terminale and the thecal sac, may become displaced, adherent, or pressed against the walls of the canal. Pressure within the sac is sufficient to cause bone erosion of the sacrum and thinning of its dorsal plate. This pressure probably explains the symptoms. Tarlov does not believe that these cysts communicate with the subarachnoid space. However, certain other authors[1,2,3,8,9,12] report diagnosing them by myelography as well as by plain roentgenography. An enlargement and "scalloping" of the sacral canal as seen on the plain roentgenograms have been described. However, a tumor arising in the sacral canal or in the substance of the sacral body (chordoma) may cause confusion.

Cysts may be single or multiple. They are non-neoplastic and rarely show any round cell inflammatory infiltration. The symptoms may be intermittent or even progressive. Usually the second or third sacral roots are involved but the condition has been encountered upon all of the sacral as well as the coccygeal roots. This latter has been suggested as a factor in the production of coccygodynia.

Abbott and his co-workers[1] reported 9 cases and reviewed 28 others. They con-

* These studies were assisted by the Departments of Anatomy and Photography, New York State School of Medicine, Syracuse, New York.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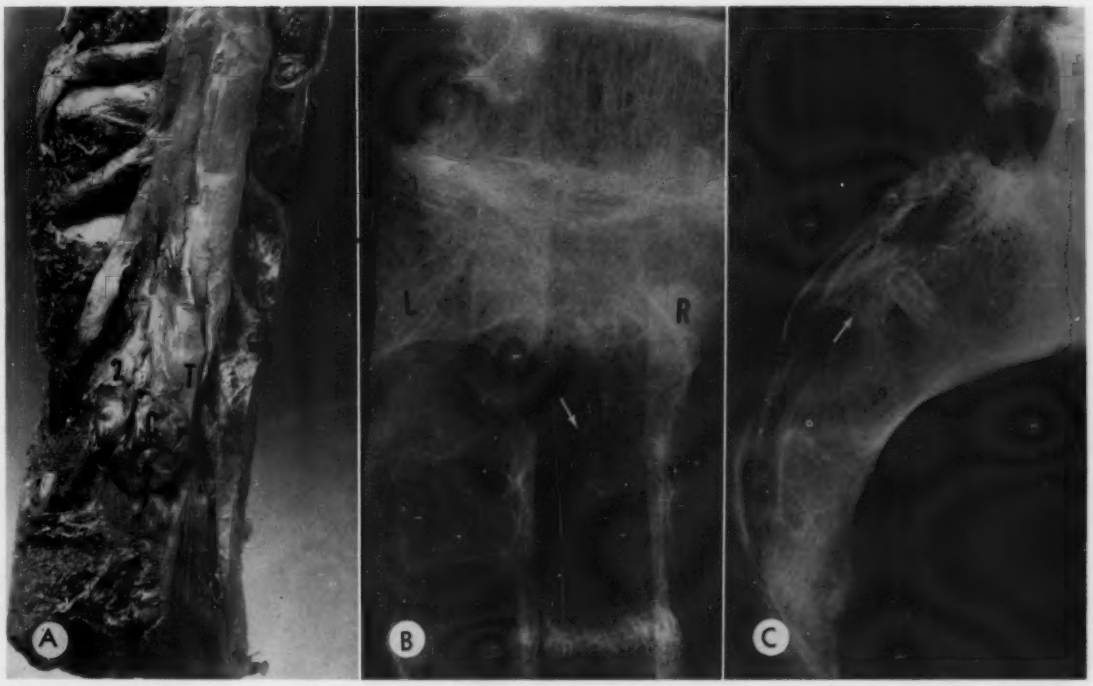

Fig. 2. (*A*, *B* and *C*) Sacrum from a seventy-four year old male. After making the roentgenograms (*B* and *C*), the posterior wall of the spinal canal was removed exposing a large lobulated perineurial cyst (C on *A*) on the third left sacral root. This did not communicate with the dural canal but lay between the left second sacral root (2) and the tip of the theca (T), both of which structures are distorted and displaced by the cyst. The third nerve lies in the anterior wall of the cyst. The bone erosion (*B* and *C*), limited by the white curvilinear lines (white arrows), has thinned the dorsal plate of the sacral canal.

cluded that the condition might be asymptomatic in certain patients but that the etiologic role played by perineurial sacral cysts in sciatic and sacrococcygeal syndromes was now well established and should be considered in low back or sciatic pain with sensory or motor deficits of the sacral root distribution.

Discussion

Sacral cysts are relatively common. They may present the clinical picture of nerve root pressure compatible with herniated disk. While some of them may be visualized by myelography, others do not communicate with the subarachnoid space. For this reason these latter will not be

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Fig. 1. (*A*) Photograph and (*B* and *C*) roentgenograms of the sectioned sacrum from a seventy-six year old female. Solitary perineurial cyst on right third sacral nerve root displaces the second sacral nerve root laterally. It does not communicate with the dural canal. The ganglion and the fibers of the third root form a part of the medial wall of the cyst (white arrow on *A*). This cyst has eroded a smooth walled spherical cavity in the second sacral segment. The cortex of this cavity, which must have formed slowly, appears on *B* and *C* as a white curvilinear line (white arrows). In these and other roentgenograms the black arrows are used exclusively to indicate the normal curvilinear lines of the sacrum. (1) Surface of pedicle forming margin of anterior foramen; (2) walls of the nerve root canals; (3) posterior foramen; (4) junction of first and second sacral segments; (5) ala of the sacrum.

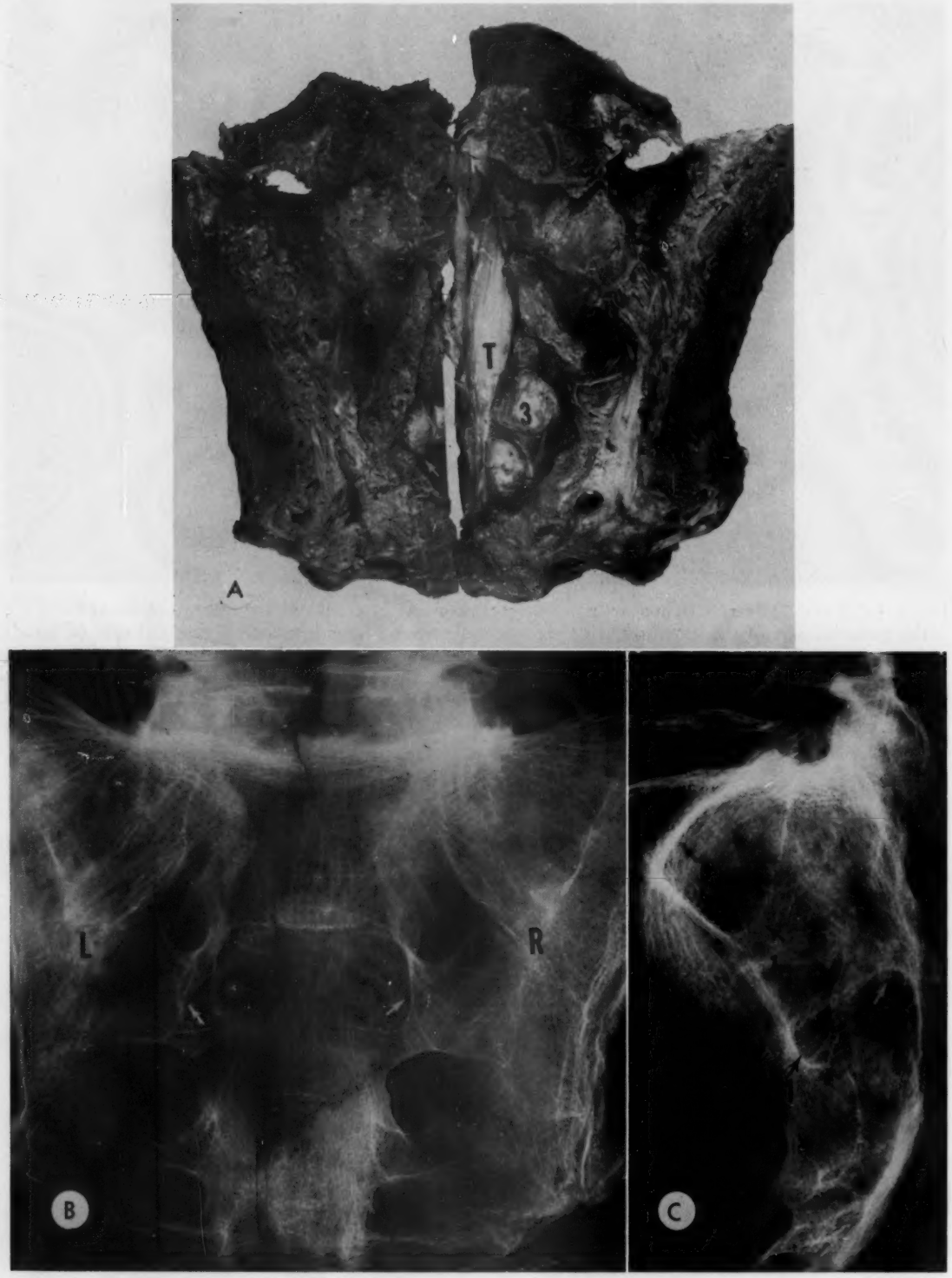

Fig. 3. (*A*) Photograph and (*B* and *C*) roentgenograms of cadaver speciman showing an example of multiple perineurial cysts. All are outside of the theca (T in *A*) which reaches downward to the S2 segment. The third left root is cystic with bony erosion laterally (white arrow on *A*). There are cysts on the second, third and fourth roots on the right side. The third root and ganglion on the right form the medial wall of the largest cyst (3 on *A*) which has eroded the sacral canal laterally and posteriorly (white arrows on *B* and *C*).

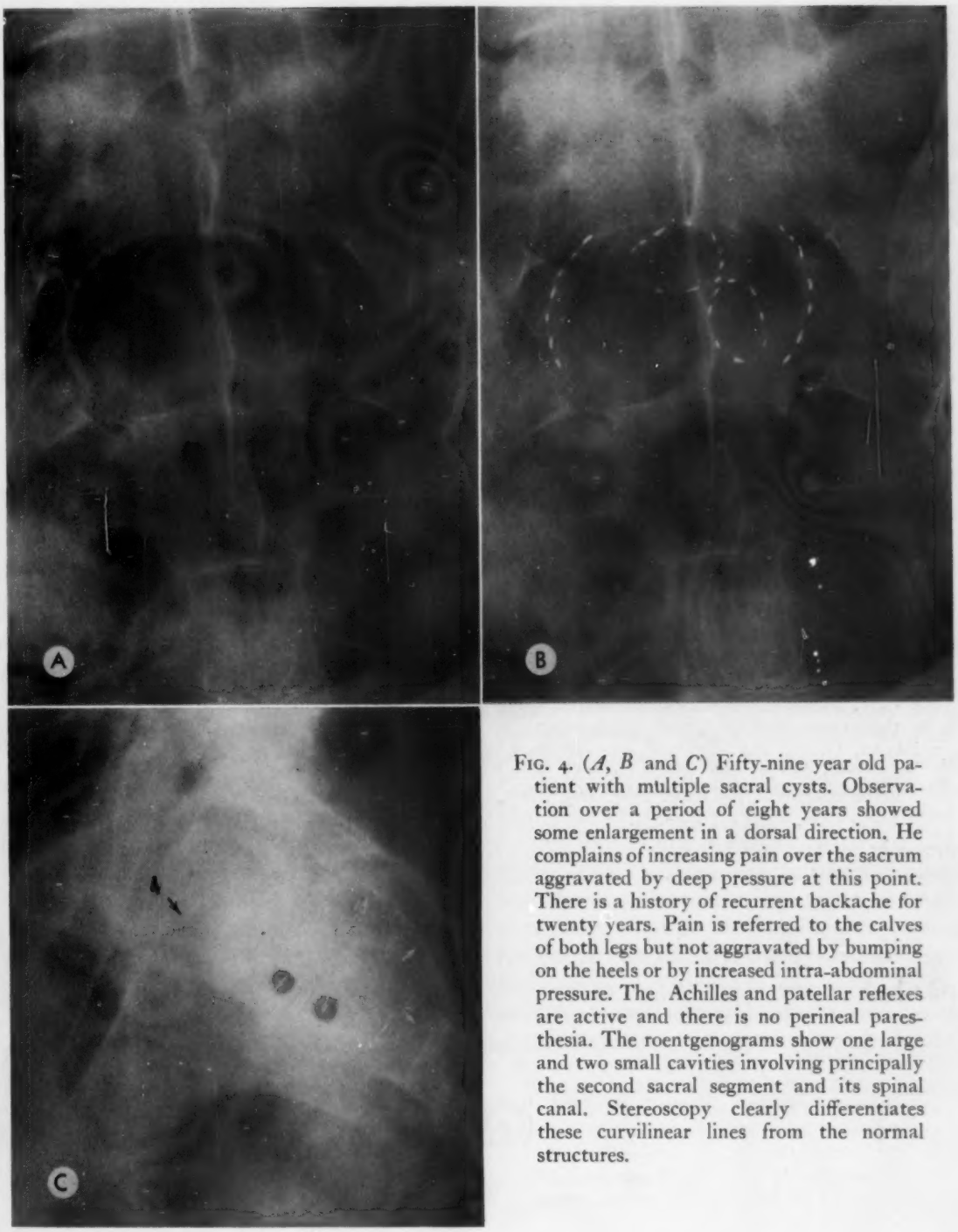

Fig. 4. (*A*, *B* and *C*) Fifty-nine year old patient with multiple sacral cysts. Observation over a period of eight years showed some enlargement in a dorsal direction. He complains of increasing pain over the sacrum aggravated by deep pressure at this point. There is a history of recurrent backache for twenty years. Pain is referred to the calves of both legs but not aggravated by bumping on the heels or by increased intra-abdominal pressure. The Achilles and patellar reflexes are active and there is no perineal paresthesia. The roentgenograms show one large and two small cavities involving principally the second sacral segment and its spinal canal. Stereoscopy clearly differentiates these curvilinear lines from the normal structures.

shown by the contrast media and it becomes desirable to seek on plain roentgenograms the white curvilinear line which limits the slowly enlarging cyst cavity. Good stereoscopic studies are essential (Fig. 4, *A* and *B*), care being taken to identify and avoid confusion with the normally curved lines in this region. Since

perineurial sacral canal cysts develop slowly, the areas of bony erosion were sharply delineated by a white curvilinear line in each of the three cadaver specimens examined (Fig. 1, *B* and *C;* Fig. 2, *B* and *C;* and Fig. 3, *B* and *C*). This marking must not be confused with the normal sacral cortical markings of similar contour. In the lateral projection these are the alae of the sacrum and the lateral masses of the various segments. In the posteroanterior projection, the foramina, the nerve root canals, the margins of the vertebral body segments or even gas shadows may cause confusion.

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REFERENCES

1. Abbott, K. H., Retter, R. H., and Leimbach, W. H. Role of perineurial sacral cysts in sciatica and sacrococcygeal syndromes; review of literature and report of 9 cases. *J. Neurosurg.*, 1957, *14*, 5–21.
2. Archer, V. W., Cooper, G., Jr., and Cimmino, C. V. Occult meningocele of sacrum; report of three cases. *Radiology*, 1948, *51*, 691–695.
3. Baker, G. S., and Webb, J. H. Intrasacral meningocele causing backache and sacral nerve pain: report of case. *Proc. Staff Meet., Mayo Clin.*, 1952, *27*, 231–234.
4. Hyndman, O. R., and Gerber, W. F. Spinal extradural cysts, congenital and acquired; report of cases. *J. Neurosurg.*, 1946, *3*, 474–486.
5. Jacobs, L. G., Smith, J. K., and Van Horn, P. S. Myelographic demonstration of cysts of spinal membranes. *Radiology*, 1954, *62*, 215–221.
6. Rexed, B. A., and Wennström, K. G. Arachnoidal proliferation and cystic formation in spinal nerve-root pouches of man. *J. Neurosurg.*, 1959, *16*, 73–84.
7. Schreiber, F., and Haddard, B. Lumbar and sacral cysts causing pain. *J. Neurosurg.*, 1951, *8*, 504–509.
8. Schurr, P. H. Sacral extradural cyst: uncommon cause of low back pain. *J. Bone & Joint Surg.*, 1955, *37-B*, 601–605.
9. Seaman, W. B., and Furlow, L. T. Myelographic appearance of sacral cysts. *J. Neurosurg.*, 1956, *13*, 88–94.
10. Strully, K. J. Meningeal diverticula of sacral nerve roots (perineurial cyst). *J.A.M.A.*, 1956, *161*, 1147–1152.
11. Strully, K. J., and Heiser, S. Lumbar and sacral cysts of meningeal origin. *Radiology*, 1954, *62*, 544–549.
12. Taheri, Z. E., Riemenschneider, P. A., and Ecker, A. Myelographic diagnosis of sacral perineurial cyst. *J. Neurosurg.*, 1952, *9*, 93–95.
13. Tarlov, I. M. Sacral Nerve-Root Cysts: Another Cause of the Sciatic or Cauda Equina Syndrome. Charles C Thomas, Publisher, Springfield, Ill., 1953.
14. Weiford, E. C. Sacral perineurial cysts, with case report. *Cleveland Clin. Quart.*, 1950, *17*, 106–111.

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FRACTIONATED X-IRRADIATION OF THE MAMMALIAN EMBRYO AND CONGENITAL ANOMALIES*

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THERE are three methods by which x-irradiation can produce exencephalia or herniated brain in the mouse fetus.[44,45] Mature sperm may be x-irradiated to a high level, and the offspring carried beyond the first generation when such congenital anomalies occasionally appear. The mechanism here is genetic. A second method is by x-irradiation of the embryo at the time of most active neurogenesis when there are abundant, highly radiosensitive neuroblasts and cranial osteoblasts. The destruction of these cells, along with the inducting mechanism of the prechordal plate, results in failure of the cranial roof to close and then follows an eversion of the damaged foreparts of the embryonic brain. The mechanism here is direct damage to the inducting centers and the differentiating cells. The third method is by x-irradiation of the very early embryo at any time prior to or during implantation, even prior to the first cleavage of the egg. The mechanism here again is to disrupt the processes of normal development by damaging chromosomes in the earliest progenitors of all embryonic cells. The end result, the exencephalous fetus, appears to be the same no matter which of the three x-irradiation insults is employed.† There are many radiomimetic agents which can also elicit this congenital anomaly[44,45] so that exencephalia is not an anomaly peculiar to x-irradiation insult but rather this condition reflects the sensitivity of the developing organism to any interfering agent. However, in this report we are concerned principally with the relation of ionizing radiations to the incidence of this congenital anomaly in the mouse, *particularly when the exposure is fractionated.*

Accepting 50 r as the minimum level of single exposure which could produce exencephalia (although 25 r caused 33 per cent of 0.5 day embryos to die *in utero*), it seemed important to determine whether 50 r, fractionated into two equal 25 r exposures at various times between 0.5 and 8.5 days postconception, might be *more* or *less* tolerable as measured both by resorption and the incidence of exencephalia. This project has involved 1387 pregnant mice and 3,598 implantation sites so that the data appear to be statistically adequate to support the conclusions of this paper.

MATERIALS AND METHOD

In a preliminary study of control (non-irradiated) pregnancies (Table 1), it was found that, while intrauterine death and resorption does occur (5.7 per cent), fetal deaths are extremely rare and cerebral anomalies, such as exencephalia, were *never found* in 630 implantations. The report[22] that resorptions occur at the rate of some 12 per cent is not borne out with our CF_1 stock. This may be due to another fact; namely, that instead of counting the corpora lutea against the implantations and delivered fetuses, we begin with the implantation as evidence of successful fertilization. To include all corpora lutea is to

TABLE I

CONTROL (UNIRRADIATED) PREGNANCIES

Pregnancies	61	
Implantation Sites	630	
Implantations/Pregnancy	10.3	
Normal Embryos	591	94%
Resorptions	37	5.7%
Dead Fetuses	2	0.3%
Exencephalies	0	0.0%

† A fourth method has developed, namely, 100 r to the ovary produced exencephalia for three generations.

* From the Radiological Research Laboratory, Columbia University, New York, New York. Based on work performed under Contract AT-30-1-GEN-70 for the United States Atomic Energy Commission.

assume that every matured ovum is equally and completely fertilizable, which certainly is not justified. It is, however, the general experience that successful fertilization will lead to development at least as far as gastrulation, and death at this stage can be identified in the uterus at 17.5 days' gestation by a resorption site. Thus, the criterion of fertilization has been the vaginal plug at 9:00 A.M. following a night of mating opportunity. The total number of implantations are counted at the time of sacrifice of the gravid female at 17.5 days' gestation (shortly prior to the expected delivery). Of course, a vaginal plug is no guarantee of successful fertilization either, so that only gravid females are reported even though they may have uteri with resorptions of all embryos.

Timed matings thus have provided pregnancies that are dated correctly to within ±6 hours, since matings most generally occur late in the evening. The irradiation exposures at 0.5 day were made before 11 A.M. the morning following mating opportunity, just after isolating the females with vaginal plugs. All subsequent exposures were made during the morning hours so that the embryonic or gestation ages given are in half days, beginning with 0.5 day and continuing through 8.5 days.

All irradiated and pregnant females in this experiment were sacrificed at 17.5 days and a record made of the implantation sites, the resorptions, dead fetuses and the gross anomalies. Each fetus was bereft of its membranes and placed in fresh Bouin's fluid for subsequent study and photography. Some were prepared for serial histologic sectioning and staining.

The x-irradiation was carried out under the same geometric conditions throughout; namely, single tube at 72 cm. target distance to gravid uterus, 184 kv., 30 ma., filtration 0.28 mm. Cu and 0.50 mm. Al and dose rate 25 r/min. Each mouse was stained on its back with an innocuous red dye following x-irradiation to distinguish it from all non-irradiated mice. It was returned to its regulation mouse cage in the animal room where it was given food and water *ad libitum*. The temperature of the animal room was kept at 72° F., and the humidity was controlled.

The data were recorded with respect to (A) total implantation sites, (B) dead fetuses, (C) resorptions, (D) gross anomalies, and (E) apparent normals. Data have been kept for each litter and there has been no evidence of reduction in litter size due to the x-irradiation used.

EXPERIMENTAL DATA

The present study was indicated because of the previous findings that 50 r delivered at one time to mouse embryos between 0.5 and 9.5 days after conception resulted in either, or both, resorptions and congenital anomalies at every period studied. Table II represents a composite of the previous data. It will be seen that 50 r causes maximum intrauterine death and resorption when it is imposed at 0.5 day (before the first cleavage of the fertilized egg), and that exencephalies (congenital cerebral hernias) were produced by x-irradiation at various times even before the implantation of the embryo. Thus, it seemed obvious that one should determine whether 50 r delivered in two equally fractionated doses of 25 r, but spaced at different times

TABLE II

X-IRRADIATION OF THE EARLY MOUSE EMBRYO (DOSE 50 r)

Embryonic Age (50 r)	Total No. Implantations	Per Cent "Normal"	Per Cent Resorptions	Per Cent Exencephalies
0.5	52	58	42	0
1.5	90	95	5	0
2.5	95	73	24	3
3.5	76	88	9	3
4.5	53	92	8	0
5.5	37	77	17	6
6.5	77	92	8	0
7.5	25	96	0	4
8.5	51	96	0	4
9.5	12	90	10	0
Totals	568	85.7	12.3	2.0

in early embryonic development, might have a different effect either in quantity or in quality.

The data for the present study are derived from examinations of 387 pregnant mice and 3,598 implantations so they must be presented largely in tabular form (see Table III).

The period of study extended from 0.5 day after conception through 8.5 days. There were 36 possible combinations of the 50 r dose fractionated into two equal exposures of 25 r. There was a maximum of 20 pregnancies and 167 implantations for one pair of irradiations and a minimum of 6 with 54 implantations for another, for an average of 10.8 pregnancies and 95.5 implantations for the entire study of 36 combinations of fractionated doses. Thus, the data appear to be quantitatively sufficient to draw conclusions.

If one begins with data from a single 25 r exposure, the importance of the initial exposure at 0.5 day can be indicated. This is shown at the top of Table III in which it is obvious that even as little as 25 r at 0.5 day (before the first cleavage of the fertilized egg) causes about one-third of the implantations to result in intrauterine resorptions. However, it must be pointed out that, while combinations of 0.5 day and other days (total 50 r) rarely exceeded the value of 33 per cent for the single exposure of 25 r, this indicates biologic variability but still within a high range, resulting in an average of 25 per cent resorptions. This is so far in excess of 25 r irradiation effects at any other time that it must be taken as conclusive that an exposure of 25 r at 0.5 day after conception is indeed damaging to the mouse embryo.*

There are but few combinations of 25 r exposures in which there are no resorptions (1.5+3.5 and 4.5+8.5 days) so that, for some reason, these periods may be considered as much less vulnerable to 25 r irradiation than is 0.5 day. Likewise, there were very few dead fetuses in the entire study (total 20) and these occurred most frequently in the embryos irradiated on 0.5 day which survived early intrauterine death and resorption.

This and the two preceding studies have concentrated on the gross and easily recognized congenital anomaly of cerebral hernia or exencephaly, as an indicator of embryonic damage by x-irradiation. Even at this low exposure level it occurred in 0.5 per cent of the fetuses. There is every presumption that all embryos exposed to 25 r at any time from conception through day 8.5 at least carry some permanent effect of the exposure on their nervous system so that it is erroneous to assume that the absence of exencephaly and the gross appearance of normality are assurances of 100 per cent normality in fact.

With regard to the so-called "normal" fetuses, these would be delivered and would survive for varying periods, but there is already sufficient evidence that many, most, or possibly all are subnormal or deficit mice in one way or another. Certainly, there is stunting in fetuses that are otherwise topographically well proportioned. The degree of "normality" will have to await behavioral tests. One can state (Table IV) that 85 per cent of the mice receiving 50 r fractionated into two 25 r exposures between 0.5 and 8.5 days "appeared normal," 13 per cent were resorbed, 0.5 per cent were killed as fetuses, and 0.5 per cent were exencephalics, but such statistics would give small comfort to any pregnant human interested in the probability that her child would be "normal" after comparable exposures at comparable times (whatever they prove to be).

The summary given in Table IV, when compared with the data from single exposures of 50 r, *shows that fractionation tends to reduce slightly the incidence of exencephalia.* The total number of "apparently normal" animals is the same (85 per cent) whether the 50 r is given at one time or fractionated. Likewise, the resorption percentage is the same, so that there appears to be slight benefit of fractionation with

* It has since been found that 5 r at this time kills 9.3 per cent of the embryos.

TABLE III

X-IRRADIATION OF THE EARLY MOUSE EMBRYO (DOSE 50 r FRACTIONATED INTO 2×25 r)

Gestation Days (25 r) (25 r)	No. Pregnant Females Irradiated	No. Implantations	Average No. Litter	"Normal" Fetuses		Exencephalies		Resorptions		Dead	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
0.5	18	160	8.8	107	73	0	0	53	33	0	0.0
0.5+1.5	20	167	8.4	126	76	1	0.5	39	23	1	0.5
0.5+2.5	12	108	9.0	71	65	0	0	37	35	0	0
0.5+3.5	14	127	8.9	87	68	2	2.0	36	28	2	2.0
0.5+4.5	14	111	8.0	95	85.6	0	0	13	11.7	3	2.7
0.5+5.5	14	126	9.0	92	73	0	0	33	26	1	1
0.5+6.5	12	103	8.7	79	76	1	1	23	23	0	0
0.5+7.5	12	104	8.7	80	76	0	0	22	22	2	2
0.5+8.5	17	160	9.4	126	78.8	0	0	34	21.2	0	0
1.5+2.5	9	96	10.6	81	85	1	1	13	13	1	1
1.5+3.5	8	65	8.1	63	97	2	3	0	0	0	0
1.5+4.5	8	74	9.3	70	93	0	0	4	7	0	0
1.5+5.5	11	94	8.6	84	89.3	0	0	10	10.7	0	0
1.5+6.5	10	93	9.3	74	80	0	0	19	20	0	0
1.5+7.5	8	81	10.1	75	92.6	0	0	6	7.4	0	0
1.5+8.5	12	106	8.8	96	90.6	3	2.8	7	6.6	0	0
2.5+3.5	8	67	8.4	59	88	0	0	8	12	0	0
2.5+4.5	6	54	9.0	50	92.6	0	0	4	7.4	0	0
2.5+5.5	10	105	10.5	86	82	0	0	18	17	1	1
2.5+6.5	12	106	8.8	99	93.4	1	1	6	5.6	0	0
2.5+7.5	8	85	10.6	82	96.5	0	0	3	3.5	0	0
2.5+8.5	9	83	9.1	70	84.3	1	1.3	12	14.4	0	0
3.5+4.5	14	149	10.6	135	90.6	0	0	13	9	1	0.4
3.5+5.5	8	80	10	71	88.8	0	0	9	11.2	0	0
3.5+6.5	8	84	10.7	69	82	0	0	15	18	0	0
3.5+7.5	10	96	9.6	93	97	1	1	2	2	0	0
3.5+8.5	7	67	9.6	58	87	0	0	9	13	0	0
4.5+5.5	9	97	10.8	86	89	0	0	9	9	2	2
4.5+6.5	12	115	9.8	93	80	0	0	21	19	1	1
4.5+7.5	13	136	10.4	123	90.4	1	0.6	12	9	0	0
4.5+8.5	10	74	7.4	74	100	0	0	0	0	0	0
5.5+6.5	10	97	9.7	87	90	0	0	10	10	0	0
5.5+7.5	13	132	10.2	117	89	2	1.5	12	9	1	0.5
5.5+8.5	9	85	9.5	76	89.4	0	0	7	8.2	2	2.4
6.5+7.5	9	81	9.0	73	90	1	1.3	6	7.4	1	1.3
6.5+8.5	10	96	9.6	89	92	0	0	7	9	0	0
7.5+8.5	11	94	8.5	83	88	1	1	9	10	1	1

TABLE IV

SUMMARY

50 r FRACTIONATED (2×25 r) FROM 0.5 to 8.5 DAYS' GESTATION COMPARED WITH 50 r SINGLE EXPOSURE

	Fractionated Exposure		Single Exposure	
Total no. pregnant females irradiated	387		52	
Total no. implantations examined	3,598		625	
Average no. per pregnant female	9.3		12.02	
Total no. apparently "normal"	3,072	85.3%		85.7%
Total no. exencephalies	18	0.5%		2.0%
Total no. resorptions	488	13.0%		12.3%
Total no. dead fetuses	20	0.5%		0.0%

respect to the incidence of this particular anomaly.

There are two other ways in which these data may be analyzed. First, if all data are assembled with respect to the initial exposure to 25 r, regardless of the time of the second exposure, their importance might be indicated. This is shown in Table v. It will be seen that those embryos which received the first irradiation insult of 25 r on day 0.5 (before the first cleavage of the egg) resulted in the minimum percentage of so-called "normal" offspring and the maximum percentage of intrauterine death and resorption (23 per cent). Since these data for 0.5 day include combinations with all other days from 1.5 to 7.5, this is indeed significant. *No other day approaches 0.5 with respect to the lethality effects of x-irradiation.* The exposure which resulted in the largest percentage of "normal" and the least percentage of resorptions was on day 6.5, just after implantation and before active organogenesis. Exencephalies occurred most frequently in those embryos with initial exposure at 1.5 days and least at 4.5 days, both times showing a high proportion of so-called "normals," indicating greater tolerance of the irradiation. If one compares here (lowest line of data in Table v) the effect of single 25 r exposure at day 0.5, it is evident that the 33 per cent gross deleterious effects from 25 r are high, no matter whether or not there is an added 25 r at a

TABLE V

ANALYSIS OF INITIAL EXPOSURE EFFECT; SUMMARY: 50 r FRACTIONATED (2×25 r)*

Initial 25 r Day	No. Implantations	Average No. Litter	"Normal" Fetuses		Exencephalies		Resorptions		Dead	
			No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
0.5+	1,006	8.76	756	*75.0*	4	0.4	237	*23.0*	9	0.90
1.5+	609	9.2	543	90.0	6	*1.1*	59	7.8	1	*0.1*
2.5+	500	9.4	446	89.4	2	0.4	51	12	1	0.2
3.5+	476	10.1	424	89.0	4	0.9	48	10.1	1	0.2
4.5+	422	9.6	375	90.0	1	*0.3*	42	9.2	3	0.7
5.5+	314	9.8	280	89.5	2	0.5	29	9.0	3	*1.0*
6.5+	177	9.3	162	*91.0*	1	0.7	13	*7.7*	1	0.6
7.5+	94	8.5	83	88	1	1	9	10	1	*1.0*
0.5 only	160	8.8	107	67	0	0	53	33	0	0

* Those with similar initial exposure to 25 r are grouped together. Second exposure was any time thereafter to 8.5 days. *Note:* The high and low percentage values in each category are italicized.

TABLE VI

ANALYSIS OF CONGENITAL DEFECTS*

Day†	Total Implantations	Exencephalies		Resorptions		Deaths	
		No.	Per Cent	No.	Per Cent	No.	Per Cent
0.5	1,006	4	0.4	237	*23.5*	9	*0.90*
1.5	776	7	*0.9*	98	13.0	2	0.30
2.5	704	3	0.42	91	12.9	1	*0.14*
3.5	735	5	0.68	92	12.5	3	0.40
4.5	810	1	*0.12*	76	9.4	7	0.86
5.5	716	2	0.27	108	15.1	4	0.56
6.5	775	3	0.37	107	13.7	2	0.24
7.5	809	6	0.74	72	*8.9*	5	0.60
8.5	775	5	0.66	85	10.9	3	0.40

* These composite data are for all embryos receiving 25 r on a specific day of development from 0.5 to 8.5 days. The other 25 r of the 50 r fractionated exposure was given at various times between 0.5 and 8.5 days. This table points up the most critical period following 25 r for the three abnormal conditions, when in combination with any other day at 25 r.

† This column gives the day on which 25 r was given to the embryo, but the additional 25 r was given to the same embryo in all possible combinations of days.

Note: The high and low percentage values in each category are italicized.

Maximum incidence: Exencephaly after exposure at 1.5 days; resorption after exposure at 0.5 days; intrauterine deaths after exposure at 0.5 and 4.5 days.

subsequent embryonic stage.

A second analysis may be made by accumulating all data relating to exposure on any single day, regardless of whether it was the initial exposure. This is shown in Table VI. Here again it is evident that every exposure of 25 r at 0.5 day is likely to result in at least 23 per cent resorptions, for the data are identical in the two tables. However, for all other days the data will be different, as indicated by the total number of implantations listed. Intrauterine death and resorption occur less frequently the later the embryo is exposed, either to the initial 25 r or to a subsequent 25 r. The production of exencephalia is essentially the same, occurring most frequently when exposure is at 1.5 days and least often at 4.5 days, whether it is the initial or the subsequent exposure. Thus, we must admit that exencephaly is most likely to occur if irradiation is imposed at 1.5 days after conception.

Such a study of daily fractionations naturally leads to a study of shorter interval fractionations, namely, on an hourly basis. The data from 44 pregnancies and 376 implantations with hourly intervals between the 25 r exposures on day 8.5 are shown in Table VII. The day 8.5 was chosen because it was more likely to produce exencephalies. Here a maximum interval of 6 hours between exposures would make the embryos $8\frac{3}{4}$ days of age.

The data indicate that the percentage of exencephalies is reduced in every combination of fractionations below that of 4 per cent, observed when there was a single exposure of 50 r (see Table II for comparison). The shorter interval fractionations naturally produced some exencephalies while in the 5 and 6 hour fractionations there were none. Thus, there appeared to be some advantage to the embryo by fractionation beyond 4 hours with respect to the incidence of this anomaly. The fact that resorptions occurred on day 8.5 to an average of 8.2 per cent of all implantations can be explained solely on the basis of normal expectancy of 5.7 per cent as determined from control studies (see Table I). However, short term fractions gave higher resorption and fetal death values. This may mean that the attempts of the developing organism at scavenging the dead cells and repair are deleteriously affected by super-

TABLE VII

FRACTIONATION EFFECTS: MOUSE EMBRYO

(8.5 days + 50 r divided into 2×25 r; hourly intervals)

Interval	No. Pregnant Females	No. Implantations	"Normal" Fetuses		Exencephalies		Resorptions		Dead		Stunted Fetuses	
			No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1 hour	11	88	78	88	1	1	7	8	2	3	0	
2 hours	11	81	68	85	2	2	10	12	0		1	1
3 hours	5	49	46	94	0		2	4	0		1	2
4 hours	6	55	48	87	1	2	6	11	0		0	
5 hours	8	71	68	95	0		3	5	0		0	
6 hours	3	32	29	90.6	0		3	9.4	0		0	
Average Totals and Percentages	44	376	337	89	4	1.6	31	8.2	2	0.6	2	0.6
Controls 8.5 days—50 r single exposure	3	25	23	92	2	8	0		0		0	

imposing a second exposure within 1–4 hours. Certainly the percentage of "apparently normal" fetuses following a single exposure of 50 r is 96 per cent and that of short term fractionation data is 89 per cent. All fractionations in such short intervals are more harmful to the embryo than the single exposure.

Attention is now directed to the study of whole litters and to the gross appearance of anomalies following fractionation exposures of the mouse embryo (Plates I, II and III, Fig. 1–24). The photographs illustrate the position within the bicornuate uterus of embryos with exencephalia (Fig. 1, 3, 5, 17, 18 and 20) following different combinations of 50 r fractionated into two 25 r doses. Since this gross cerebral anomaly occurred in every possible combination of fractionated irradiations, but in varying positions within the litter, one must suspect individual differences in response to radiation insult. These may be related to the genotype but more likely to some slight difference in development, such as the timing of changes pertinent to the closure of the neural folds and the overdeveloping of the cranial roof. Since most of these irradiations occurred prior to implantation and organogenesis, one must admit that the damage probably interfered with gastrulation and cephalization through chromosomal effects.

In examining these illustrations one must not forget that *the frequency of this anomaly, exencephalia, is relatively very low* and that resorptions occur much more frequently, especially when the fractionated x-irradiation occurs at an early stage of embryonic development. Figure 9, for instance, shows complete resorption of all implantations (10) in one mouse, while Figure 10 shows a single fetus of a large litter in which the only observable effect was a rather superficial evidence of hemorrhage. However, in Figure 7 one sees resorption of all embryos except one, and that one developed exencephalia.

When one observes these gross anomalies of exencephalia, the question always arises as to whether the herniated tissue may be in fact nervous tissue. Thus, Figure 11

PLATE 1

FIG. 1. Entire litter at 18.5 days following exposure to 25 r at 0.5 day and 25 r at 3.5 days after conception. Note single exencephaly. Litter mates appear normal but are slightly reduced in size.

FIG. 2. Two members from another litter exposed to 25 r at 0.5 day and 25 r at 3.5 days. Note reduction in head of member with exencephaly. Posterior structures of exencephalous member appear normal.

shows a gross protrusion from the head of a fetus whose litter mates (Fig. 12) appeared normal. When this tissue was sectioned and studied, it was seen to be very definitely of nervous origin. Figure 13 shows ependyma with sloughed off cells in the neurocoele, Figures 14 and 15 show choroid plexus, and Figure 16 a formed but unidentifiable nervous body, all in the mass extruded from the head. Likewise, sections of the head of this fetus (with protrusion removed) show a very much reduced and damaged brain (Fig. 22–24) at different levels. Nevertheless, much of the bony structure of the head appears quite normal except for the cranial roof which has failed to close over.

DISCUSSION

Since the two previous papers in this series[44,45] included a discussion of the various method of induction of neurologic teratologies in the mouse, that phase is omitted in the present study. However, brief reference is made to the effect of fractionation on the adult with which we may compare the effects on the embryo.

While the current study deals with the mouse embryo, it is appropriate that brief mention be made of earlier studies which pointed the way to the present interest in neurologic teratologies, if for no other reason than to alert us to the possible causes. In 1948 Trampusch[58] produced microcephaly and acephaly by x-irradiating urodele embryos as early as the blastula stage. The yolk plugs persisted and exogastrulation often followed. It was suggested that the anomalies may not have been due to neural sensitivity but rather to effects on the mesodermal substratum, especially the prechordal plate. The head inductions arise in the prechordal plate and roentgen rays interfere with the morphogenetic movements in this gradient field. This interpretation is still the most probable. Pleurodeles gastrulae were irradiated and this suppressed the competence of the ectoderm without affecting the dorsal lip of the blastopore.[61] Rugh[46] described the histologic damage to the central nervous system of developing amphibian larvae (tadpoles), showing the sloughing off of neuroblasts into the neurocoeles.

One of the earliest studies of x-irradiation effects on neurogenesis was by Schneller [51] who showed that to damage the chick embryo's neuroblasts the x-irradiation must be direct, that shielding of the embryo would only cause death through effects on the extra-embryonic blood vascular system. Since then, it has been stated that the nervous system of the developing chick embryo was its most radiosensitive system.[43] The developing nervous system of birds is especially sensitive to CO_2, ultrasonation and the combination of the two, so that morphogenetic movements which normally close the neural tube are affected, resulting in cephalic anomalies.[29–31]

Following early studies on x-irradiation of the rodent embryo by Russell and Rus-

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Fig. 3. Total dose of 50 r fractionated to 25 r at 1.5 day and 25 r at 2.5 days after conception, as found in two uterine horns. Note single exencephaly and slight variations in size among litter mates.

Fig. 4. Entire litter (2) showing exencephaly in both members, following 25 r at 1.5 day and 25 r at 3.5 days, both prior to implantation.

Fig. 5. Entire litter as found in 2 uterine horns showing two exencephalies out of a total of 12 members, and others showing various degrees of stunting. Exposures were 25 r at 1.5 day and 25 r at 8.5 days.

Fig. 6. Total dose of 50 r fractionated to 25 r on day 2.5 and 25 r on day 6.5 (after implantation). Note exencephaly and slight size variations among litter mates (partial litter).

Fig. 7. Total dose of 50 r fractionated at 25 r on day 2.5 and 25 r on day 8.5 (after implantation). In this instance all implanted embryos were resorbed except one and that developed exencephaly, as shown.

Fig. 8. Total dose of 50 r fractionated into two equal exposures of 25 r on days 2.5 and 5.5. One resorbed and one dead fetus, with slight size variations among other litter mates, resulted.

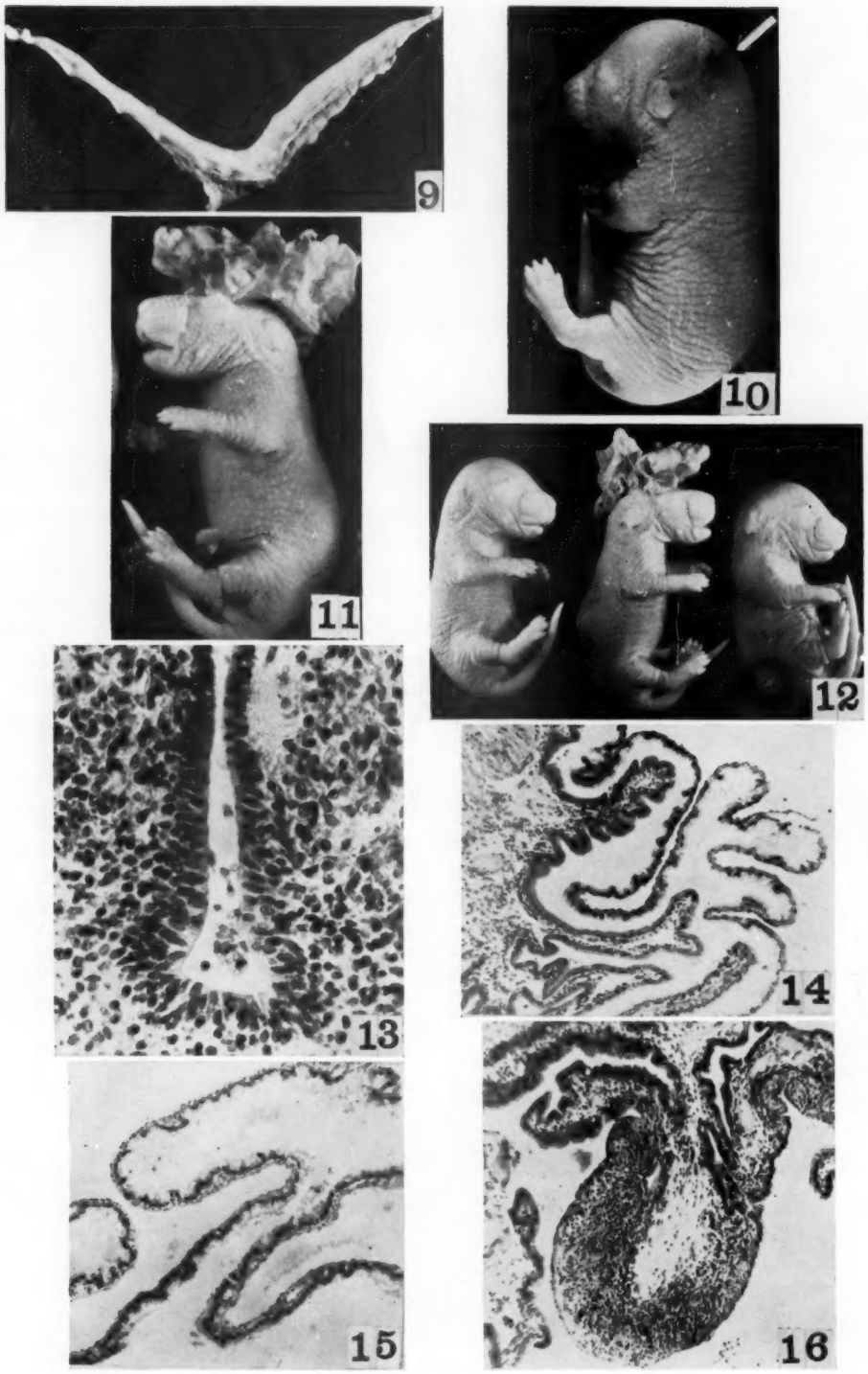

PLATE II

FIG. 9. Uterine horns of mouse exposed to 50 r fractionated to 25 r on day 3.5 and 25 r on day 6.5. Photograph shows complete resorption of all implanted embryos (10) at 18.5 days' gestation. (Reproduced by permission from *Military Medicine*.)

sell[48–50] and by Hicks,[17–19] there appeared a rather thorough analysis of mal-development of the mouse embryonic nervous system following x-irradiation and other insults.[34] This was followed by a fertile suggestion of using radiation as a tool in mammalian developmental neurology.[20] In all such studies it is important that one not lose sight of the fact that the entire embryo is x-irradiated, and that there are certainly effects on all tissues and organs, even though the effects on the central nervous system may be the most graphic and easily demonstrated. It has been shown[5,63] that developmental (congenital) anomalies could be produced only by direct irradiation of the embryo, and that shielding the embryo and irradiating the mother did not cause the same effects. In fact, maternal exposure, even though it might affect the metabolism of the mother, did not affect the embryo.

Fraser and Hall[12] have supported the idea of the Russells of "critical periods" of radiosensitivity for various organ systems, and noted the development of facial vibrissae in the mouse after using doses of 250–350 r at 8 to 14 days. It is suggested[52] that shifts in the sex ratio of mice might be due to gamma irradiation of the embryos. Rugh and Jackson[47] found that there was a reversal of the expected radiosensitivity of the fetal gonads, the presumptive testes being more affected than the presumptive ovaries. A study is in progress on all body tissues and organs of irradiated embryos and while this paper deals with effects on neurogenesis, it is not presumed that this is an exclusive or necessarily even a primary or major effect. A recent study by Murakami and Kameyama[35] again emphasizes the central nervous system as the most obviously affected organ system, describing meningoceles, pseudoencephaly, and, incidentally, spina bifida. Further, we must not forget that the act of birth does not distinguish between a period of early radiosensitivity and later radioresistance, for in almost all mammalia the embryo is born prematurely. This was shown by irradiating white rats from birth to 14 days and finding the cerebellum particularly vulnerable.[27]

It is also important to avoid the impression that such congenital anomalies as we describe can be brought about solely by ionizing radiations. When pregnant mice were put under conditions simulating those of high altitudes[22] where the environment was anoxic, there developed anencephaly and cranioschisis, particularly when the gestation period was 8.5 days. Giroud and Martinet[13] gave 50,000 units of vitamin A orally (in oil) to rats at 8–10 days gestation and produced anencephaly involving protrusions of the prosencephalon and mesencephalon of the brain.* If pregnant white rats are given vitamin A and then are exposed to roentgen rays, the result is that more anophthalmia and microphthalmia

* This had been shown first by Cohlan.[7]

Fig. 10. Total exposure of the pregnant mouse was 50 r on days 3.5 and 6.5, fractionated into two equal doses of 25 r. Single member of litter showed only evidence of hemorrhage of the head.

Fig. 11. Single embryo from litter exposed to total dose of 50 r fractionated to 25 r on days 4.5 and 7.5.Note marked protrusion of brain hernia. Slight evidence of eye is present. (Reproduced by permission from *Military Medicine* and *Annual Review of Nuclear Science.*)

Fig. 12. Same exencephalous embryo as in Figure 11 with two litter mates to show size relationships, and right side view of anomaly. Note that structures posterior to the head appear to be quite normal. (Reproduced by permission from *Military Medicine* and *Acta Embryologica.*)

Fig. 13–16. These are four microscopic sections of the protrusions shown in Figure 11, all of which give evidence of neural tissue. Figure 13 shows the ependyma with sloughed off cells in the neurocoele, and in Figure 14 and 15 the choroid plexus is definitely shown. The formed body in Figure 16 could not be identified. All of these structures developed outside of the cranium. The irradiation was prior to the active neurogenesis.

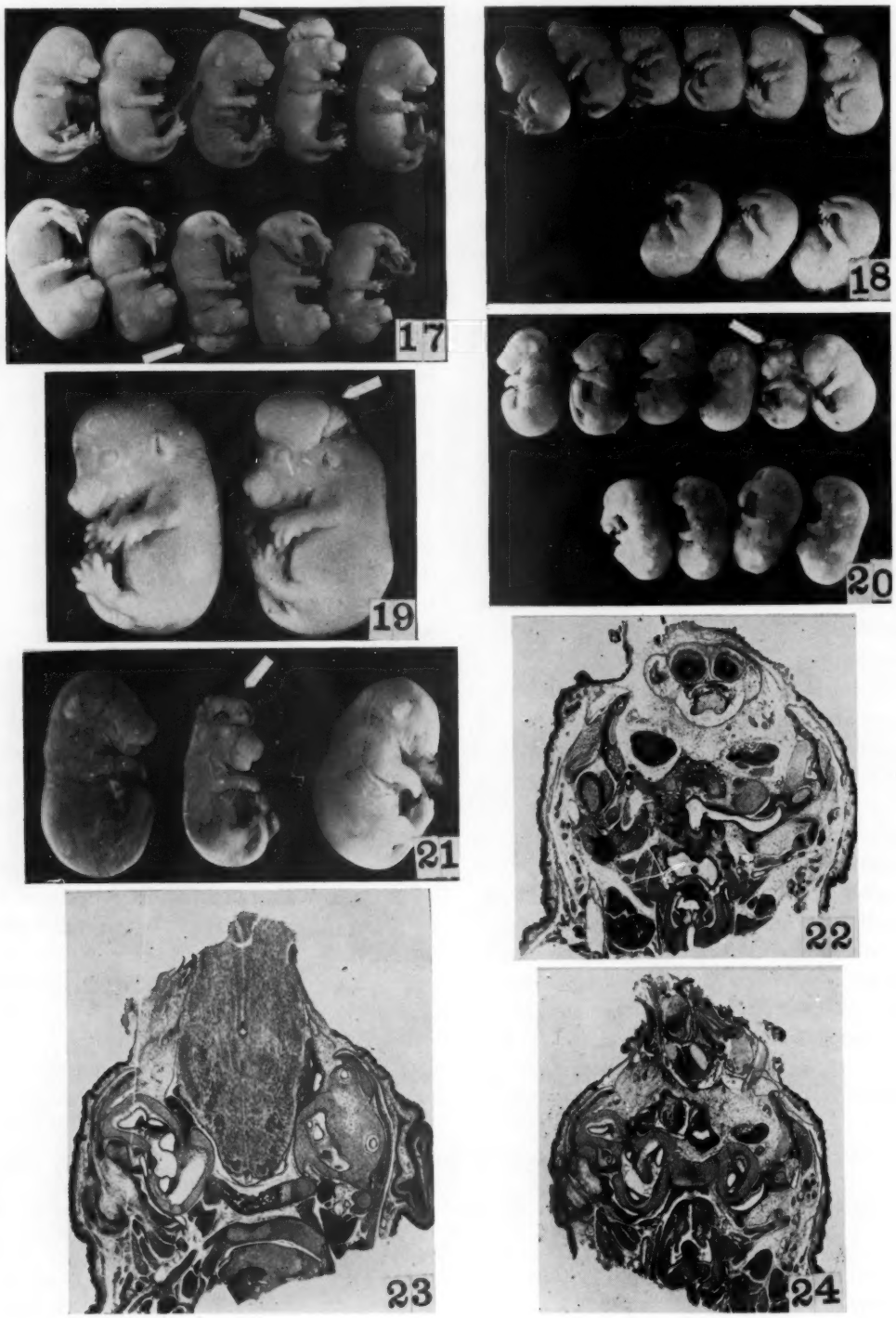

PLATE III

FIG. 17. Entire litter which was exposed at 5.5 days to 25 r and at 7.5 days to an additional 25 r, for a total dose of 50 r. Two exencephalies were produced. These are shown in the same position as found within the uterine horns.

FIG. 18. Entire litter exposed to 50 r fractionated into two equal doses of 25 r on days 6.5 and 7.5, both

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are produced than in those having irradiation alone.[66] There are, in fact, many radiomimetic compounds which seem to either cause chromosome breaks or mutations, resulting in the same types of anomalies that irradiation alone will cause.[3] The important consideration is that the embryo at any stage is a very sensitive, dynamically changing organism which is subject to almost any insult. While it appears to have considerable recuperative power, particularly toward maintaining topographic integrity, insult during or before the stages of tissue and organ formation may result in deficit embryos, fetuses and delivered newborn. Their survival will depend in part on the degree of the reorganization ability of the undamaged parts.

It is of interest that one of the first explanations of anencephaly and rachischisis posterior was based upon a study of a human embryo of 18 mm.[56] The failure of the neural tube to close was said to be secondary to a deficiency of the mesoderm which underlies the ectoderm. These severe brain anomalies may therefore be due not so much to neural damage but to failure of the cranial vault to enclose it properly. Patten[41] has reviewed the various causes of teratologies in the human and enumerates various developmental factors such as too little or too much growth, too little or too much resorption of temporary structures, resorption normal but in the wrong place or growth processes normal but in the wrong place. There is certainly no single common denominator to congenital defects unless one could emphasize the importance of *timing* as the correlative influence in normal embryonic development. The *integration* of the various developmental changes in any embryo is probably the most important and the least understood factor, and one which depends so much on timing. This is most graphically shown in normal embryology in the development of the optic equipment from head ectoderm and outgrowing brain ectoderm, converging to form the well proportioned eye.

The record of human cerebral anomalies is incomplete, and the causal relations have not been established, with few exceptions. Worcester *et al.*[65] studied over 29,000 births at the Boston Lying in Hospital and found 2.3 per cent grossly malformed. Of the stillbirths 79 per cent had defects referable to the central nervous system. Gruenwald[14] stated that human "anencephaly and myeloschisis are essentially the same type of malformation resulting from simple arrest of development—the neural tube fails to close." Such an arrest of development might be caused by lack of oxygen or glucose due to transitory disturbances in the placental bed[6] and is most likely to be damaging during the first 16 weeks of pregnancy.[42] It is believed[62] that the ability of the skull to mold properly decreases rapidly after the thirty-sixth week in the human and that anencephaly results from a profound disturbance of development of the most anterior part of the neural tube and adjacent structures which are to be enclosed by the skull. Unglaub[60] tends to emphasize ionizing radiations (among other things) as particularly responsible for central nervous system congenital malformations and states, "Germinal injur-

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after implantation but before active neurogenesis. A single cerebral anomaly of exencephaly is seen.

Fig. 19. Part of Figure 18 is enlarged to show better the exencephalous embryo.

Fig. 20. Entire litter exposed to 50 r, fractionated to 25 r each on days 7.5 and 8.5, the latter exposure at the beginning of active neurogenesis. Note single exencephaly among litter mates of varying sizes.

Fig. 21. Three members from litter exposed in a manner similar to that shown in Figure 20.

Fig. 22–24. Microscopic sections through brain (after removal of protruded portion of brain) of exencephalous fetus shown in Figure 11, Plate II. Note almost complete absence of brain tissue in the head. The neural tissue that can be identified is abnormal.

ies are more important than hereditary factors in the etiology of congenital malformations."* Some investigators[28] believe that roentgen rays may cause microcephalus, hemicephalus, hydrocephalus or mongolism, and found over a five year period an incidence of 2.73 per cent of malformations. It is suggested that anoxia, due to emotional stress, might be a causative factor, as well as German measles during the first trimester. Anderson *et al.*[1] studied 37,585 births in which 1.6/1,000 were anencephalics and 2.3/1,000 had deformities of the central nervous system. Curiously, they found that as the social status of the parents fell there was a steady increase in the incidence of anencephalia among the stillbirths with a maximum of 3.1/1,000 in Glasgow.

It is stated that there were mild changes in the fetal nervous system but absolutely no macroscopic injuries to a human embryo exposed to 3,000 r (to pelvis) during the second and third month of pregnancy.[2] One would like to know more accurately the delivered dose, the exact age of the embryo at each irradiation, and the neurologic development of the child before and during adolescence. The overwhelming evidence, both from controlled animal experimentation and the majority of reports of human embryonic exposures, supports the thesis that much greater caution should be exercised when the embryo, particularly during the first trimester, might be x-irradiated in the course of either diagnosis or therapy. This statement is underscored when we point out that the embryonic gonads, the carriers of hereditary influences, begin to develop at an early stage and are also subject to changes in the form of gene mutations which can be permanent in surviving progeny.

Except for one report,[4] the embryo has not been used in determining the effect of fractionated x-irradiation until this current study. Auerbach[4] found that a fractionated dose resulted in greater damage to the developing eyes and lumbosacral skeleton than did the same dose delivered in one period. While the incidence of malformations was not increased, the severity of the defects was greater under fractionation. The fractionation interval was 30 minutes, but in embryonic development this might be critical.

The theory of fractionation is based largely upon three premises: *First*, that if there is a critical phase in the response mechanism, by fractionation one is more likely to hit it even with a lower exposure. The *second* is that the period of greatest sensitivity is during active mitosis and that since the intermitotic period is generally the longer, one is more apt to hit the particular cells during mitosis if bombardment is in two or more shots rather than one, with a suitable interval between. The *third* concept is that whatever damage is inflicted upon the cells, tissues, organs or organism, if the dose is divided to allow an interval without irradiation, there is opportunity for rest and recovery from the ill effects. The result is that more irradiation can be tolerated. In tumor therapy fractionation is generally the practice so that the surrounding normal tissue is not seriously damaged by the lower single exposures and the highly mitotic tumor tissue can be more frequently bombarded. There are other factors relating to advantages of fractionated irradiation of tumors which do not concern us here. There are, however, so many variables in dose delivery that one must be certain that all but one are stabilized when fractionation effects are studied. For instance, there is the specific cell, tissue, organ or organism concerned, which varies tremendously; the type of ionizing radiation and its energy; the total dose delivered; the number and magnitude of the fractions; the intervals between fractions; and the total period of observation.[55] In this study of fractionation exposure of the embryo, we have limited the time of observation to the early gestation period and

* Wesley[61] states: "X-rays have caused a 6 per cent increase in congenital malformations in the United States in the last 30 years."

have had but one variable, the interval between equal fractions. However, as is demonstrated in this paper, there is actually at least another variable, which is being studied, and that is the response of the embryo at different developmental stages; *i.e.*, at the various combinations of fractionation time. The organisms are identical (within the strain); the radiations are invariable; the total dose is 50 r; the rate is 25 r/min.; the time of delivery is 1 minute for the fractions of 25 r; but the period between fractions varies with 36 possible combinations within the span of the first 8.5 days of gestation.

A brief review of pertinent papers on fractionation studies is appropriate here before evaluating the findings of this study. Pack and Quimby[36] believed that the critical interval for fractionation was somehow related to the life span of the cells involved and that recovery during the latent period varied for different tissues. For skin erythema it appeared to be about 4 hours. Paterson and co-authors[37–39] referred to radioresistant phases and felt that by fractionating the dose one would be more apt to hit the tissues sometime during radiosensitive phases. Further studies were made on the differences in the recovery processes from fractionated whole body irradiation delivered over definite over-all periods of time with a constant dose rate. The maximum extension of time was three weeks for mice. A half-recovery time of 7.4 days was established where two fractions of varying intervals were used. It seemed that the ability of the tissue to recover is impaired with the extension of the over-all time of irradiation. The authors believe that the fractionation experiments suggest interference with a single physiologic system or with several systems operating as one.

Henshaw[15] reduced the daily dose from 25 r to 5 r and was able to extend the life of the mice from 20 to 36 weeks. Ellinger[10] extended the life of mice by fractionation of the lowest single lethal dose and stated that the effect depends upon the size of the single fraction as well as the over-all exposure time, but of the two the size of the fraction is the more important.[11] The lethal dose was determined for mice[39] and it was found that by fractionating the exposures over 20 days the LD_{50} could be increased from 518 r to 950 r. Likewise, mice were given total body irradiation and it was found that the LD_{50} was 486 r for single exposure, 542 r for 2 daily fractions, 678 r for 4 daily fractions, and 850 r for 8 daily fractions.[24] Further increase of the intervals between exposures increased the LD_{50} values still further. It was also found that the life expectancy was reduced more with a single exposure than with fractionated exposures of the same total dose.[21] Kereiakes and his co-workers[25,26] found that mice survived better with the greater fractionation intervals, and that within 5 hours after exposure there appeared to be considerable recovery of nucleic acid metabolism in the small intestines and of general mitosis.

Comprehensive studies on time-dose relations have appeared in which both the size of the fraction as well as the type of tissue are emphasized.[8,9] If fractionation exposures occurred in periods when no recovery was allowed, then there was no advantage of fractionation. The so-called radiosensitive and radioresistant periods seemed to be related to the mitotic index of the tissues studied. Therapy for tumors would be improved by optimum timing of individual treatments, and the fact that supralethal doses may be administered may be related to an impairment of the host resistance following previous exposures. Du Sault and her co-workers stated, "An increased differential effect in tumor and normal tissue, therefore, can be obtained by this timing of treatments."[9]

Any study dealing with fractionation of exposure to ionizing radiations must consider the rather new concepts of recovery and increased tolerance. It was found that 518 r whole body exposure was the LD_{50} for monkeys if it is given in a single session but if protracted over 20 days the dose

must be increased to 946 r.[40] The recovery was attributed in part to the higher mean red blood cell count, even though in some isolated cases the count of survivors was as low as that of moribund monkeys. Mole[32,33] has emphasized that "The killing efficiency per roentgen of a limited exposure is much greater than for continued irradiation because so much of the continued irradiation is wasted." He also believed that limited exposures had an irreversible effect which was not found so characteristic of chronic irradiation. Recovery from a single exposure was found to be affected by other doses, given before or afterwards. He stated that damage caused by radiation was linearly proportional to dose and was additive and that death was bound to follow when accumulated radiation reached a particular level, though it might take some days or weeks after this moment before death actually occurred. Sproul[54] estimated that mice exposed to 400 rad of neutrons or 595 rad of gamma rays demonstrated a one-half recovery time of 4 days. The rate of recovery was definitely affected by the size of the dose. Kallman[23] found that at least two factors; namely, the intestinal mucosa and the bone marrow, *each had its own recovery rate*, suggesting a differential recovery rate of the various tissues in whole body irradiation of any organism. This must apply also to the developing embryonic tissues.

Fractionation studies with early embryonic development present situations somewhat different from those one faces in irradiating adult tissues. However, the differences may be in degree as well as in quality if one first admits that the end result of embryonic damage may be a teratology and the end result of exposure of adult tissue is its death. Certainly, fractionation separated by days is different from fractionation with intervals of an hour or more, and this difference is more obvious in embryonic than in adult fields of exposure, due to the high level of mitotic and differentiational activity of the embryonic cells. Since the percentage of apparently "normal" individuals and of resorptions appears to be about the same, whether there is a single 50 r exposure at any time during the first 8.5 days of development of the mouse, or the dose is given in two equal fractions of 25 r at any combination, there is no advantage of one method over the other. However, if one is concerned primarily with the production of gross, surviving cerebral anomalies such as exencephalia, then a single exposure of 50 r appears to be slightly more productive than fractionation. When one considers the possibility that some of the 85 per cent surviving "apparently normal" fetuses may be in fact subnormal by various functional and long-term survival tests, then from a purely social point of view there is little to choose between the two methods. The exencephalic cannot survive long after delivery, but its litter mate called "normal" may long survive and in the case of humans even become a public burden.

When one reviews the findings of these studies, it seems obvious that the only logical over-all conclusion is that, while the embryo can often survive the devastations of ionizing radiations and can re-form its body from residual apparently unaffected cells, it may in fact be a deficit organism following any exposure prior to the completion of organogenesis. The deficit may be anencephaly, microcephaly, microphthalmia, or even a functional deficit, measurable only as a lowered intelligence quotient. There is no presumption that other organ systems of irradiated embryos escape similar deficits from x-irradiation, so further studies in this direction are imperative. The degree of deficiency may be difficult to establish, and, due to biologic variability, may never be satisfactorily quantitated. However, if and when mouse data may be extrapolated to the human, it may be necessary to tell any woman in early pregnancy, who should have diagnostic irradiation, that she risks the chance (small that it may be) of having her embryo killed, or developing a dead fetus, or one with a severe cerebral anom-

aly, or having a surviving child with unmeasurable deficiencies. If she points to the 10 per cent of children now being born with abnormalities as a normal human situation and within the range of normal expectancy, and insists upon her right to continue within that possibility, the doctor will at least have done his minimum duty. In the meantime, it is possible for the medical and dental professions to reduce the incidence of embryonic irradiation to those relatively few cases where the mother's life is actually in danger if diagnostic data are not made available, and possibly even where therapy is indicated. But to continue to add to the sum total of human malformed, no matter how minor the damage by irradiation, can no longer be condoned.

SUMMARY AND CONCLUSIONS

1. Pure strain CF_1 Swiss white mice were time-mated to provide 387 dated pregnancies resulting in 3,598 implantations. These were x-irradiated to 25 r twice during the embryonic period from 0.5 to 8.5 days after conception in order to test the effect of 50 r fractionated x-irradiation on the early mammalian embryo. There were 36 combinations of the fractionated exposures within this period, in none of which were less than 54 implantations studied.

2. Litter size averaged 9.3 so that fertility was within the range of normality for this strain. The number of implantations was determined at 17.5 days because instances of intrauterine death and resorption could be counted then, and the other had no opportunity of devouring the abnormal newborn, which is their common practice. This is considered a more accurate method of acquiring data than by balancing corpora lutea against total implantations since unfertilizable and unfertilized eggs are not included in these statistics. In unirradiated controls 61 pregnancies gave 630 implantations which resulted in 5.7 per cent resorptions, 0.3 per cent fetal deaths, no exencephalies or cerebral anomalies, and 94 per cent normal fetuses.

3. The results following 50 r single exposure of the early embryo *or* 50 r fractionated into two equal exposures at least 24 hours and up to 7.5 days apart did not differ from each other with respect to resorptions, or total percentage which "appeared to be normal." However, *with fractionation there were fewer exencephalies* (brain hernias) *and also fewer fetal deaths.*

4. While exencephaly did not occur frequently after 50 r x-irradiation, it did occur in litters which had been irradiated to 25 r *on any day* from 0.5 to 8.5, regardless of when the other fraction of 25 r was delivered. Day 7.5 may be considered particularly vulnerable. Days 3.5 and 4.5 appeared to be the least vulnerable with any combination, a time just before implantation.

5. Intrauterine death and early resorptions followed most frequently x-irradiations which occurred on day 0.5, and least when the initial exposure of 25 r was on day 6.5. In general, embryonic death followed very early exposure and anomalies followed later exposures, whether single or fractionated. With single exposures of 50 r on day 0.5 (after fertilization but before the initial cleavage), there were 42 per cent deaths and resorptions but when the initial 25 r fraction was delivered at this time, the value was 23 per cent. In either case there was a very considerable increase over the 5.7 per cent found in the controls.

6. When the fractionation interval between the 25 r exposures was reduced from days to hours, it was shown that if the interval was 4 hours or less there was essentially no difference between 50 r single or 50 r fractionated into 2×25 r. When the intervals were of 5–6 hours, there appeared to be slightly less over-all damage to the embryos, with fewer exencephalies, deaths, and stuntings.

7. While a few litters showed 100 per cent anomalies after 50 r fractionated irradiation and many had no obvious anomalies following identical irradiation, the percentage values must be emphasized

rather than the isolated cases. The disparities may be due to normal biologic variability, to genotypic differences, or to varying abilities and degrees of topographic "recovery." The tendency to regard as "normal" all fetuses which exhibit no gross anomaly is ruled out in this analysis. It is presumed that any litter in which a gross cerebral anomaly, such as exencephalia, has occurred as a result of x-irradiation cannot escape some damage to each of its members. This is further supported by the fact that 50 r fractionated before the completion of organogenesis killed an average of 14 per cent of the embryos, an increase of some 9 per cent over the controls.

8. The concept of a "critical period," which has been used in experimental embryology for half a century, cannot be applied to the radiosensitivity of the nervous system since it has been demonstrated that the brain can be affected by exposure to roentgen rays at any time before its development has been completed, even before the initial cleavage of the fertilized egg.

9. It is apparent, from this study, that the mouse embryo is radioresponsive in that congenital anomalies can be produced by roentgen rays from the time of conception at least until organogenesis is completed. Intrauterine deaths may develop following even very low level irradiations. The anomalies following early exposure are probably due to chromosomal aberrations and, following exposure during organogenesis, are probably due to disruptions in the morphogenetic movements of differentiation resulting from the killing of vulnerable cells. While the embryo has remarkable powers of topographic reorganization, it is still to be proved whether it can survive irradiation insult without some permanent damage. It is contended that x-irradiation is an insult which results in a deficit embryo, fetus, newborn, and surviving adult when it is applied at any time prior to the completion of organogenesis.

10. Extrapolation of radiation effects from the mouse embryo to the human is not valid. However, such findings suggest that not only should the embryo and fetus be protected against unnecessary x-irradiation, but that fractionation of dose may reduce the incidence of some anomalies, but not the over-all damage to the embryo.

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REFERENCES

1. ANDERSON, W. J. R., BAIRD, D., and THOMSON, A. M. Epidemiology of stillbirths and infant deaths due to congenital manformations. *Lancet*, 1958, *1*, 1304; also, *Obst. & Gynec. Survey*, 1958, *13*, 835–836.
2. ANDRISKA, J., FRIGYESI, G., KISZELY, G., and NAGY, M. Die Wirkung einer grossen Dosis Röntgenstrahlen auf die Entwicklung eines menschlichen Foetus. *Strahlentherapie*, 1958, *105*, 630–638.
3. AUERBACH, C. Radiomimetic substances. *Radiation Res.*, 1958, *9*, 33–47.
4. AUERBACH, R. Effects of single and fractionated doses of x-rays on mouse embryos. *Nature*, London, 1956, *177*, 574.
5. BRENT, R. L. Indirect effect of irradiation on rat embryos. *A.M.A. J. Dis. Child.*, 1957, *93*, 7–8.
6. BUCHNER, F. Human congenital malformations in light of modern pathology. *German Med. Monthly*, 1956, *1*, 261; also *Obst. & Gynec. Survey*, 1957, *12*, 380–382.
7. COHLAN, S. Q. Excessive intake of vitamin A as cause of congenital anomalies in rat. *Science*, 1953, *117*, 535–536.
8. DU SAULT, L. Time-dose relationships. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *75*, 597–606.
9. DU SAULT, L. A., EYLER, W. R., and BURNS, W. M. Studies of time-dose relationships; effect of fractionation. *Radiology*, 1958, *71*, 709–715.
10. ELLINGER, F. Influence of dose fractionation on lethal x-ray effect produced by total body irradiation in mice. *Radiology*, 1947, *49*, 238–241.
11. ELLINGER, F., and BARNETT, J. C. Further studies on influence of dose fractionation on lethal x-ray effect produced by total body ir-

radiations in mice. *Radiology*, 1950, *54*, 90–92.
12. Fraser, A. S., and Hall, R. J. Effects of irradiation on foetal development. *Austral. J. Biol. Sc.*, 1958, *11*, 425–433.
13. Giroud, A., and Martinet, M. Morphogénèse de l'anencéphalie. *Arch. d'anat. micr.*, Paris, 1957, *46*, 247–264.
14. Gruenwald, P. Prematurity, congenital malformations and birth injury. Proc. Conf. Ass. for Aid of Crippled Children, New York City, 1953, pp. 160–174.
15. Henshaw, P. S. Effects of repeated small doses of x-rays on blood picture, tissue morphology, and life span in mice. *J. Nat. Cancer Inst.*, 1954, *4*, 513–522.
16. Hicks, S. P. Acute necrosis and malformations of developing mammalian brain caused by x-ray. *Proc. Soc. Exper. Biol. & Med.*, 1950, *75*, 485–489; also, 1952, *80*, 15–18.
17. Hicks, S. P. Some effects of ionizing radiation and metabolic inhibition on developing mammalian nervous system. *J. Pediat.*, 1952, *40*, 489–513.
18. Hicks, S. P. Developmental brain metabolism. *A.M.A. Arch. Path.*, 1953, *55*, 302–327.
19. Hicks, S. P. Mechanism of radiation anencephaly, anophthalmia, and pituitary anomalies. *A.M.A. Arch. Path.*, 1954, *57*, 363–378.
20. Hicks, S. P. Radiation as experimental tool in mammalian developmental neurology. *Physiol. Rev.*, 1958, *38*, 337–356.
21. Hursh, J. B., Van Slyke, F., and Casarett, G. Effect of single vs. divided x-irradiation on survival of rats. UR-317, 1954.
22. Ingalls, T. H. Prematurity, congenital malformations and birth injury. Proc. Conf. Ass. for Aid of Crippled Children, New York City, 1953, pp. 122–136.
23. Kallman, R. F. Effect of dose rate on killing efficiency and mode of death of mice by whole body irradiation. *Radiation Res.*, 1959, *9*, 137 (abstr.).
24. Kaplan, H. S., and Brown, M. B. Mortality of mice after total body irradiation as influenced by alterations in total dose, fractionation and periodicity of treatment. *J. Nat. Cancer Inst.*, 1952, *12*, 765–775.
25. Kereiakes, J. G., Parr, W. H., and Krebs, A. T. Role of certain organs in fractionated total body x-irradiation studies. AD-125586, 1956.
26. Kereiakes, J. G., Parr, W. H., and Krebs, A. T. Fractionated dose effects on survival and organ weights in x-irradiated mice. *Am. J. Physiol.*, 1957, *191*, 131–134.
27. Kosmarskaya, E. N., and Barashnew, Y. I. Effect of single irradiation by x-rays on developing brain of rats. *Med. Radiol.*, 1958, *3*, 23–31.
28. Lewin, H., and Fischer, G. Über kongenitale Missbildungen. *Zentralbl. Gynäk.*, 1958, *80*, 413–429.
29. Lutz, H. Malformations par action du gas carbonique et des ultra-sons chez l'embryon de poulet. XV International Congress Zoology, Sect. VII, paper no. 10.
30. Lutz, H., and Lepy, M. Action du gas carbonique sur le blastoderme non incubé d'oiseau. *Bull. Soc. Zool. de France*, 1958, *83*, 76–81.
31. Lutz, H., and Lutz-Ostertag, Y. Acervencéphalie expérimentale in vitro. *J. Embryol. & Exper. Morphol.*, 1958, *6*, 504–509.
32. Mole, R. H. On wasted radiation and the interpretation of experiments with chronic irradiation. *J. Nat. Cancer Inst.*, 1955, *15*, 907–914.
33. Mole, R. H. Quantitative observations on recovery from whole body irradiation in mice. II. Recovery during and after daily irradiation. *Brit. J. Radiol.*, 1957, *30*, 40–46.
34. Murakami, U. Clinico-genetic study of hereditary disorders of nervous system, especially problems of phenogenesis. *Folia Psychiat. et Neurol. Japonica*, 1957, Suppl. 1.
35. Murakami, U., and Kameyama, Y. Effects of low dose x-radiation on mouse embryo. *A.M.A. J. Dis. Child.*, 1958, *96*, 272–277.
36. Pack, G. T., and Quimby, E. H. Time intensity factor in irradiation. Am. J. Roentgenol. & Rad. Therapy, 1932, *28*, 650–667.
37. Paterson, E. Time-intensity factor in x-ray irradiation. *Brit. J. Radiol.*, 1944, *17*, 26–30.
38. Paterson, E., and Thompson, M. V. Time-intensity factor in x-irradiation. II. Influence of dosage rate. *Brit. J. Radiol.*, 1948, *21*, 414–419.
39. Paterson, E., Gilbert, C. W., and Matthews, J. Time intensity factors and whole body irradiation. *Brit. J. Radiol.*, 1952, *25*, 427–433.
40. Paterson, E. Factors influencing recovery after whole-body radiation. *J. Fac. Radiologists*, 1954, *5*, 189–199.
41. Patten, B. M. Varying developmental mechanisms in teratology. *Pediatrics*, 1957, *19*, 734–748.
42. Pitt, D. Congenital malformations and maternal rubella. *Australian M. J.*, 1957, *1*, 233; also, *Obst. & Gynec. Survey*, 1957, *12*, 522–524.
43. Reyss-Brion, M. La sensibilité différentielle de certaines ébauches de l'embryon de poulet aux rayon x, à différents stades du développement. *Arch. anat. micr.*, Paris, 1956, *45*, 342–353.
44. Rugh, R., and Grupp, E. X-irradiation exencephaly. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *81*, 1026–1052.
45. Rugh, R., and Grupp, E. Exencephalia following x-irradiation of pre-implantation mammalian embryo. *J. Neuropath. & Exp. Neurology*, 1959, *18*, 468–481.
46. Rugh, R. Histological effects on embryo follow-

ing x-irradiation. *J. Morphol.*, 1949, *88*, 483–501.

47. Rugh, R., and Jackson, S. Effect of fetal irradiation upon subsequent fertility of offspring. *J. Exper. Zool.*, 1958, *138*, 209–222.
48. Russell, L. B. X-ray induced developmental abnormalities in mouse and their use in analysis of embryological patterns. I. External and gross visceral changes. *J. Exper. Zool.*, 1950, *114*, 545–602.
49. Russell, L. B., and Russell, W. L. Effect of irradiation on pre-implantation stages of mouse embryo. *Anat. Rec.*, 1950, *108*, 521.
50. Russell, L. B., and Russell, W. L. Analysis of changing radiation response of developing mouse embryo. *J. Cell. & Comp. Physiol.*, 1954, *43*, 103–149; also, *J. Exper. Zool.*, 1956, *131*, 329–395.
51. Schneller, S. M. B. Mode of action of hard x-rays on 33- and 60 hour chick embryo. *J. Morphol.*, 1951, *89*, 367–396.
52. Shapiro, F. B., and Shapiro, N. I. Variation in sex ratio in mice as possible result of gamma irradiation of their embryos. *Doklady Akad. Nauk. SSSR*, 1958, *122*, 215–225.
53. Shiraki, H., Uchimura, Y., Matsuoka, S., Miyake, M., Takeya, S., Tamagawa, C., Koyano, K., Amano, S., Araki, M., Ayres, W. W., and Haymaker, W. Effects of atomic radiation on brain in man. *J. Neuropath.*, 1958, *17*, 79–137.
54. Sproul, J. A. Estimates of recovery in mice exposed to neutrons and gamma rays. *Radiation Res.*, 1958, *9*, 187 (abstr.).
55. Storaasli, J. P., Rosenberg, S., Krohmer, J. S., and Friedell, H. L. Effect of fractionation on lethal properties of total body radiation in rats. NYO-4919, 1955.
56. Stroer, W. F. H., and van der Zwam, A. Anencephaly and rachischisis posterior with description of human hemicephalus of 18 mm. *J. Anat.*, 1939, *73*, 441–450.
57. Trampusch, H. A. L. Studies on defective development following x-ray radiation. II. Point of attack and its mechanics. *Acta Neerlandica Morph.*, 1949, *6*, 336–352.
58. Trampusch, H. A. L. Studies on defective development following x-ray radiation. *Acta Neerlandica Morph.*, 1948, *6*, 43–58.
59. Trampusch, H. A. L. Action of x-rays on morphogenetic field. *Koninkl. Ned. Akad. Wetenschap. Proc.*, 1958, *61C*, 530–545.
60. Unglaub, I. Zu den Ursachen angeborener morphologischer Störungen des Zentralnervensystems. *Zentralbl. Gynäk.*, 1957, *79*, 873–898.
61. Wesley, J. P. Background radiation as cause of fatal congenital malformation. *Int. J. Rad. Biol.*, 1960, *2*, 97–118.
62. Williams, R. Skull at birth. *J. Fac. Radiologists*, 1957, *8*, 290–311.
63. Wilson, J. G., and Karr, J. W. Effects of irradiation on embryonic development. I. X-rays on 10th day of gestation in rat. *Am. J. Anat.*, 1951, *88*, 1–34.
64. Wolff, É., and Reyss-Brion, M. L'effet des rayons x sur la gastrula d'un Batracien urodèle: Pleurodeles waltlii. *Compt. rend. Acad. sc.*, 1957, *244*, 3172–3175.
65. Worcester, J., Stevenson, S. S., and Rice, R. G. 677 congenitally malformed infants and associated gestational characteristics. *Pediatrics*, 1950, *6*, 37–50; also, 208–222.
66. Woollam, D. H., Pratt, C. W., and Fozzard, J. A. Influence of vitamins upon some teratogenic effects of radiation. *Brit. M. J.*, 1957, *1*, 1219–1221.

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Sixty-first Annual Meeting: Ambassador Hotel, Atlantic City, N. J., September 27–30, 1960.

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Forty-third Annual Meeting: Broadmoor Hotel, Colorado Springs, Colo., May 11–14, 1961.

* *Ex officio.*

EDITORIAL

THE SIXTY-FIRST ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Sixty-first Annual Meeting of the American Roentgen Ray Society will be held at the Ambassador Hotel, Atlantic City, New Jersey, on September 27 to 30, inclusive, 1960. This year's meeting is enthusiastically anticipated as the Society has not met in this excellent convention city since 1947. The Chairman of the Committee on Local Arrangements is Dr. Robert A. Bradley who served in this same capacity in 1947. Mrs. Robert A. Bradley is Chairman of the Ladies' Committee. Since there are so few members of the American Roentgen Ray Society living in Atlantic City, Dr. and Mrs. Bradley have enlisted the aid of members of the medical profession who have had broad experience in making arrangements for the many medical meetings held in their city.

The number of commercial exhibits promises to be the largest in the Society's history according to Dr. James C. Cook, Manager of the Annual Meeting, and Mr. Clifford L. Sherratt, Honorary Member. These will be set up on the Sun Porch, in the Rotunda and in the Lounge of the Ambassador Hotel. They will be open on the first three days (Tuesday, Wednesday and Thursday) from 8:30 A.M. to 5:30 P.M. and on the fourth day (Friday) until 1 P.M.

The Scientific Exhibits Committee, of which Dr. Ted Leigh is the Chairman, reports that the exhibits this year will be of the highest order and most instructive. These will be set up on the Sun Porch and in the Venetian Room. They will be open daily at 8:00 A.M. On Tuesday, September 27, they will remain open until 8:00 P.M., on Wednesday until 9 P.M., and on Thursday until 7:30 P.M.

Registration will begin on Sunday, September 25, at 2:00 P.M. On Monday through Thursday the registration desk will be open from 8:00 A.M. until 5 P.M., and on Friday from 8:00 A.M. until 1:30 P.M.

The Annual Meeting of the Society will begin formally at 8:30 A.M. on Tuesday, September 27, in the Renaissance Room of the Ambassador Hotel. The Address of Welcome will be given by Dr. David B. Allman of Atlantic City, past President of the American Medical Association, immediately following the Call to Order by President Edward B. D. Neuhauser. The Chairman of the Executive Council, Dr. John F. Holt, will assist Dr. Neuhauser in the installation of President-Elect, Dr. Harold G. Reineke.

The Program Committee has selected forty-three papers for presentation during the four-day meeting. A wide variety of subjects will be discussed by many outstanding radiologists.

On Tuesday evening, September 27, the annual Caldwell Lecture will be given in the Renaissance Room at 8:15 P.M. This year's lecturer is Dr. Traian Leucutia of Detroit. Dr. Leucutia is well known to the members of the Society for his scholarly attainments and his many years of devoted service as Associate Editor of the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE. He will be introduced by Dr. Lawrence Reynolds, past President of our Society and Editor of the JOURNAL.

The Director of Instructional Courses, Dr. Harold O. Peterson, has again arranged an excellent group of courses to be given by a fine faculty. The courses will be held on Tuesday, Wednesday and Thurs-

day from 3:00 P.M. to 4:30 P.M. and on Friday from 1:30 P.M. to 3 P.M. on the first floor of the Ambassador Hotel where ample space is available. Details of the courses will be published in the August issue of the JOURNAL. Members and guests will be expected to fill out application blanks for the courses they plan to attend and will receive tickets in advance of the meeting.

The Annual Banquet will be held on Thursday, September 29, in the Renaissance Room at 7:30 P.M., preceded by a cocktail party at 6:30 P.M. in the Embassy Room. Excellent entertainment is being provided, followed by dancing until 1 A.M.

Pre-convention activities include important committee meetings on Saturday and Sunday, September 24 and 25. The Annual Golf Tournament for members and guests will be held on Monday, September 26, at Seaview, an excellent Golf Club just outside Atlantic City. Dr. John Fetter of Philadelphia is Chairman of the Tournament. Bus transportation will be provided from the hotel to the Club and return. At the annual golf dinner after the Tournament, prizes will be awarded to the winning players.

The Society extends a cordial invitation to all radiologists, their wives and guests to attend this meeting.

HAROLD G. REINEKE, M.D.
President-Elect
927 Carew Tower
Cincinnati 2, Ohio

PRELIMINARY PROGRAM

SIXTY-FIRST ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

The Sixty-first Annual Meeting of the American Roentgen Ray Society on September 27 to September 30, 1960, will be held at the Ambassador Hotel, Atlantic City, New Jersey.

The Executive Council will meet on Sunday, September 25.

The Seaview Golf Club will be the site of the Annual Golf Tournament for the Willis F. Manges Trophy for the Eighteen Hole Medal Handicap and the Exhibitors' Trophy for the Eighteen Hole Gross Score on Monday, September 26. After the tournament, the two trophies and also other prizes will be awarded at the Annual Golf Dinner.

This year's Caldwell Lecture will be delivered on Tuesday evening, September 27, by Dr. Traian Leucutia in the Renaissance Room.

The Instruction Courses will be held each afternoon, Tuesday through Friday. A detailed plan of the courses will be published in the August issue of the JOURNAL.

President-Elect Harold G. Reineke and his Committee have arranged the Scientific Program of the Meeting, which is as follows:

Tuesday, September 27, 1960
Renaissance Room

8:30 A.M. Call to Order, Sixty-first Annual Meeting, Edward B. D. Neuhauser, M.D., President. Address of Welcome: Dr. David B. Allman of Atlantic City, past President of the American Medical Association.

Installation of the President-Elect, Harold G. Reineke, M.D., Cincinnati, Ohio, by President Edward B. D. Neuhauser, M.D., Boston, Mass., and John F. Holt, M.D., Chairman of the Executive Council, Ann Arbor, Mich.

Inaugural Address: President Harold G. Reineke, M.D.

9:15 A.M.

Presiding: Harold G. Reineke, M.D.
Cincinnati, Ohio

1. Benign Angiomatous Tumors in Bone. Robert S. Sherman, M.D. (by invitation), New York, N. Y., and Daniel Wilner, M.D., Atlantic City, N. J.
2. The Occipito-Atlanto-Axial Joints in Rheumatoid Arthritis and Ankylosing Spondylitis. William Martel, M.D. (by invitation), Ann Arbor, Mich.
3. Tumoral Calcinosis—Report of Three Cases and Reviews of the Literature. Robert J. Reeves, M.D., Durham, N. C., and D. L. Barton, M.D. (by invitation), Durham, N. C.
4. Recognition and Management of Fractures of the Orbit. William T. Thorwarth, M.D. (by invitation), Philadelphia, Pa., Robert P. Barden, M.D., Philadelphia, Pa., and Thomas F. Graham, M.D. (by invitation), Philadelphia, Pa.
5. Photofluorography: A Critical Evaluation of Its Place in Hospital Radiographic Practice. George T. Wohl, M.D., Philadelphia, Pa., George C. Henney, A.B., B.S. (by invitation), Philadelphia, Pa., and Jay Wenger, M.D. (by invitation), Philadelphia, Pa.
6. Unusual Radiologic Manifestations of Longstanding Diabetes Mellitus. Martin W. Donner, M.D. (by invitation), Baltimore, Md.
7. The Roentgenological Diagnosis of Fetal Hydrops. Paul A. Bishop, M.D., Philadelphia, Pa.
8. The Roentgenologic Features of Trigeminal Nerve Neurofibromas. Colin B. Holman, M.D., Rochester, Minn., Hendrik J. Svien, M.D. (by invitation), Rochester, Minn., and Ignacio Olive, M.D. (by invitation), Mexico City, Mexico.
9. Gas in the Hepatic Vessels. Jerome Wiot, M.D. (by invitation), Cincinnati, Ohio, and Benjamin Felson, M.D., Cincinnati, Ohio.
10. Cineradiographic Studies of the Larynx Using Dionosil. James H. Scatliff, M.D. (by invitation), New Haven, Conn., John A. Kirchner, M.D. (by

invitation), New Haven, Conn., and Tevian J. Turkat, M.D. (by invitation), New Haven, Conn.

Tuesday Evening, September 27, 1960
Eight-fifteen o'clock

THE CALDWELL LECTURE

Heuristic Glimpses in the Evolution of Radiation Therapy

by

Traian Leucutia, M.D.

Director of the Division of Radiation Therapy, Harper Hospital, Detroit, Michigan, and Assistant Professor of Radiology at Wayne State University, Detroit, Michigan.

Introduction:

Lawrence Reynolds, M.D., Chief of the Department of Radiology, Harper Hospital, Detroit, Michigan, and Clinical Professor of Radiology at Wayne State University, Detroit, Michigan.

Wednesday, September, 28, 1960

8:30 A.M.

Presiding: John F. Holt, M.D.
Ann Arbor, Michigan

11. The Motor Activity of the Esophagus in Association with Esophageal Atresia. John A. Kirkpatrick (by invitation), Philadelphia, Pa.
12. The Lower Esophageal Vestibular Complex: An Anatomic-Roentgen Study. Constantino Zaino, M.D. (by invitation), New York, N. Y., Maxwell H. Poppel, M.D. (by invitation), New York, N. Y., Harold G. Jacobson, M.D., New York, N. Y., Harold Lepow, M.D. (by invitation), New York, N. Y., and Cahit H. Osturk, M.D. (by invitation), New York, N. Y.
13. Notes on a Rapid Small Bowel Examination. Joseph L. Morton, M.D., Indianapolis, Ind.
14. The Examination of Patients with Suspected Perforated Peptic Ulcer Using a Water-Soluble Opaque Medium. George Jacobson, M.D., Los Angeles, Calif., Clarence J. Berne, M.D. (by invitation), Los Angeles, Calif., Harvey I. Meyers, M.D. (by invitation), Los Angeles, Calif., and Leonard Rosoff, M.D. (by invitation), Los Angeles, Calif.
15. The Roentgenologic Diagnosis of Carcinoma of the Colon in Chronic Ulcerative Colitis. John R. Hodgson, M.D., Rochester, Minn., and William G. Sauer, M.D. (by invitation), Rochester, Minn.
16. The Application of Radioactive Cholografin in the Determination of Liver Dysfunction. J. R. McLaren, M.D. (by invitation), Atlanta, Ga., John Galambos, M.D. (by invitation), Atlanta, Ga., and H. Stephen Weens, M.D., Atlanta, Ga.
17. Early Radiologic Recognition of Pancreatic Carcinoma. Julian O. Salik, M.D. (by invitation), Baltimore, Md.
18. Operative Mesenteric Arteriography. Alexander R. Margulis, M.D. (by invitation), St. Louis, Mo., and Peter Heinbecker, M.D. (by invitation), St. Louis, Mo.
19. Transposition of the Great Vessels. Richard G. Lester, M.D. (by invitation), Minneapolis, Minn., George P. Wilson, M.D. (by invitation), Minneapolis, Minn., Lewis S. Carey, M.D. (by invitation), Minneapolis, Minn., Paul · Adams, M.D. (by invitation), Minneapolis, Minn., and Marina Yarbro, M.D. (by invitation), Minneapolis, Minn.
20. The Correlation of Coronary Artery Calcification with Clinical Evidence of Arteriosclerotic Heart Disease. Arthur Lieber, M.D. (by invitation), Minneapolis, Minn., and Joseph Jorgens, M.D. (by invitation), Minneapolis, Minn.
21. Anatomical Diagnosis of Atrial Septal Defect. Erik Carlsson, M.D. (by invitation), St. Louis, Mo.

Thursday, September 29, 1960

8:30 A.M.

Presiding: Fred O. Coe, M.D.
Washington, D. C.

22. Urethrography in the Adult Male. James W. Willwerth, M.D. (by invitation), Reading, Pa.

23. New Radiologic Signs of Carcinoma of the Ureter. Harry S. Bergman, M.D. (by invitation), New York, N. Y., R. M. Friedenberg, M.D. (by invitation), New York, N. Y., and Victor Sayegh, M.D. (by invitation), New York, N. Y.
24. Medullary Sponge Kidney. John A. Evans, M.D., New York, N. Y.
25. Vesicoureteral Reflux: Correlation of Roentgenographic and Radioisotope Methods. K. L. Krabbenhoft, M.D., Detroit, Mich., J. K. Cobb, M.D. (by invitation), Detroit, Mich., and M. Gordon, M.D. (by invitation), Detroit, Mich.
26. Renal Angiography in the Evaluation of Hypertension. Erich K. Lang, M.D. (by invitation), Baltimore, Md.
27. Arteriosclerotic Narrowing of Renal Arteries Associated with Hypertension. David A. Van Velzer, M.D. (by invitation), Houston, Texas, Curtis H. Burge, M.D., Houston, Texas, and George C. Morris, Jr., M.D. (by invitation), Houston, Texas.
28. Intangibles in the Treatment of Cancer. Eugene P. Pendergrass, M.D., Philadelphia, Pa.
29. The Pulmonary Manifestations and Complications of Leukemia. John A. Campbell, M.D., Indianapolis, Ind., Eugene C. Klatte, M.D. (by invitation), Indianapolis, Ind., and Robert J. Rohn, M.D. (by invitation), Indianapolis, Ind.
30. Intra-Costal Venography in Intra-Thoracic Neoplasm. John A. Tobin, M.D. (by invitation), Ann Arbor, Mich., William Fry, M.D. (by invitation), Ann Arbor, Mich., and Robert Singer, M.D. (by invitation), Ann Arbor, Mich.
31. Comparison of Contrast Media Based upon Central Nervous System Effects: Experimental Studies. Harry W. Fischer, M.D. (by invitation), Iowa City, Iowa, and John Eckstein, M.D. (by invitation), Iowa City, Iowa.
32. The Value of Angiography in the Diagnosis of Suprasellar and Parasellar Lesions. Norman E. Chase, M.D. (by invitation), New York, N. Y., and Juan M. Taveras, M.D., New York, N. Y.

Friday, September 30, 1960

8:30 A.M.

Presiding: Ralph M. Caulk, M.D.
Washington, D. C.

33. Radiation Pneumonitis. Bernard Roswit, M.D. (by invitation), Bronx, N. Y., and Sol M. Unger, M.D. (by invitation), Bronx, New York.
34. Radioiodine in the Diagnosis of Diminished Thyroid Function in Pediatrics. Eugene L. Saenger, M.D., Cincinnati, Ohio, Athena Y. King, M.D. (by invitation), Cincinnati, Ohio, and Mary Pettit, R.N. (by invitation), Cincinnati, Ohio.
35. Splenic Scintillation Scanning. Philip M. Johnson, M.D. (by invitation), Chapel Hill, N. C., Ernest H. Wood, M.D., Chapel Hill, N. C., and Stewart L. Mooring, M.D. (by invitation), Chapel Hill, N. C.
36. The Effect of Radiation upon Pulmonary Function. George Cooper, Jr., M.D., Charlottesville, Va., John L. Guerrant, M.D. (by invitation), Charlottesville, Va., and David Teets, M.D. (by invitation), Charlottesville, Va.
37. Treatment of Breast Cancer by Radical Mastectomy, Postoperative X-Ray and Intravenous Radiophosphorus. I. Hematological Effects. II. Actuarial Analysis of Survival Data. Glenn E. Sheline, M.D. (by invitation), San Francisco, Calif., M. D. Jones, M.D. (by invitation), San Francisco, Calif., H. Kohn, M.D. (by invitation), San Francisco, Calif., and C. Zippin, M.D. (by invitation), San Francisco, Calif.
38. Neutron Activation Analysis. L. R. James, M.D. (by invitation), Omaha, Neb., R. E. Ogborn, M.D. (by invitation), Omaha, Neb., A. L. Dunn, Ph.D. (by invitation), Omaha, Neb., A. J. Blotcky, B.S. (by invitation), Omaha, Neb., and G. F. Johnson, M.D. (by invitation), Omaha, Neb.,
39. Ureteral Obstruction in Stage iii Cancer of the Cervix Relieved by Low Intensity Radium Needle Implantation. Charles L. Martin, M.D., Dallas, Texas.
40. Preliminary Evaluation of High Dose Total Pelvis Irradiation Technique in the Treatment of Cancers of the

Uterine Cervix. Gilbert H. Fletcher, M.D., Houston, Texas, Paul M. Chan, M.D. (by invitation), Houston, Texas, and Felix N. Rutledge, M.D. (by invitation), Houston, Texas.

41. An Evaluation of Composite Filters. E. Dale Trout, B.S., D.Sc., Milwaukee, Wis., John P. Kelley, B.S. (by invitation), Milwaukee, Wis., and Arthur C. Lucas, B.S. (by invitation), Milwaukee, Wis.
42. Radium Pelvic Measurements for Treatment of Carcinoma of the Cervix and Fundus Uteri. Edwin C. Ernst, M.D., St. Louis, Mo., Edwin C. Ernst, Jr., M.D. (by invitation), St. Louis, Mo., Roland P. Ernst, M.D. (by invitation), St. Louis, Mo., Richard E. Ernst, M.D. (by invitation), St. Louis, Mo., and Marshall Cleland, Ph.D. (by invitation), St. Louis, Mo.
43. The Systemic Effects of Localized Radiation on Serum Proteins in Humans. Joseph L. Ackerman, M.D. (by invitation), Atlantic City, N. J., Joseph A. Linsk, M.D. (by invitation), Atlantic City, N. J., and Bernard J. Shuman, M.D. (by invitation), Atlantic City, N. J.

RELEASE OF STATEMENTS BY THE NATIONAL COMMITTEE ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

1. At its meeting in November, 1959, the Executive Committee agreed that the NCRP should make a statement with regard to the maximum permissible dose from television receivers. Such a statement has been prepared and voted upon by the full committee.

The following position has been adopted by the NCRP:

> During the past years members of the NCRP have investigated the emission of X-rays from television receivers. From a genetic point of view even sources of minute radiation are of significance if they affect a large number of people. X-rays emitted by home television sets are, therefore, of interest because of the high percentage of the population involved. In order to insure that the television contribution to the population gonad dose will be only a small fraction of that due to natural background radiation, the NCRP recommends that the exposure dose rate at any readily accessible point 5 cm. from the surface of any home television receiver shall not exceed .5 mr per hour under normal operating conditions.
>
> Laboratory and field measurements[1] have shown that with this maximum permissible exposure level the television contribution to the gonad dose at the usual viewing distances will be considerably less than 5 per cent of that due to the average natural background radiation. Most of the present television receivers already meet this requirement with a high factor of safety. In general, therefore, no changes in shielding of existing sets will be required. However, the recommended limit will insure that future television receivers, operating at higher voltages, will not contribute significantly to the population gonad dose.

[1] X-ray emission from television sets. Carl B. Braestrup and Richard T. Mooney. *Science*, Oct. 23, 1959, *130*, 1071–1074.

2. In its statements of January 8, 1957 and April 15, 1958, the NCRP specified a permissible dose of 10 (N-18 rems) to the skin of the whole body for radiation workers. It has subsequently been realized that the use of the age-proration formula for the skin of the whole body is not practical under some conditions. Therefore, the Committee will discontinue this statement and in its place make the following recommendation:

> Skin of the whole body:
>
> The maximum permissible dose to the skin of the whole body shall not exceed 30 rems per year and the dose in any 13 consecutive weeks shall not exceed 10 rems.

This statement is essentially the same as in the 1959 ICRP report except that the ICRP specified a 13-week limitation of 8 rems. It is felt that this is unnecessarily low if the annual limit is 30 rems.

3. The above changes will be incorporated in the appropriate reports of the NCRP as the case arises.

SOCIETY PROCEEDINGS AND NEWS ITEMS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Ambassador Hotel, Atlantic City, N. J., Sept. 27–30, 1960.

American Radium Society
Secretary, Dr. Charles G. Stetson, 350 Engle Street, Englewood, N. J. Annual meeting: Broadmoor Hotel, Colorado Springs, Colo., May 11–14, 1961.

Radiological Society of North America
Acting Secretary-Treasurer, Dr. Robert P. Barden, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: December 4–9, 1960, Cincinnati, Ohio.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Chicago, Ill., Feb., 8–11, 1961.

Section on Radiology, American Medical Association
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: June 13–17, 1960, Miami Beach, Fla.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building. Rochester, Minn. The Fall 1960 examination will be held in Cincinnati, December 10 through 13. The deadline for filing applications is July 1, 1960. A Special Examination in Nuclear Medicine (for diplomates in Radiology or Therapeutic Radiology) will be offered, provided there are sufficient applications.

Tenth International Congress of Radiology
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

Seventh Inter-American Congress of Radiology
Counselor for the United States, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colorado. The meeting will be held in São Paulo, Brazil, September 3–10, 1961.
Secretary-General, Dr. Walter Bomfim-Pontes, Rua Cesario Motta, No. 112, São Paulo.

Alabama Radiological Society
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

American Nuclear Society
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

Arizona Radiological Society
Secretary, Dr. R. Lee Foster, 1313 N. Second St., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. J. B. Scruggs, Arkansas Baptist Hospital, Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary, Dr. Paul Riemenschneider, Medical College. State University of New York, Syracuse, N. Y. Annual meeting to be announced.

Atlanta Radiological Society
Secretary, Dr. Wilson T. Edenfield, 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

Blockly Radiological Society
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St. Philadelphia, Pa.

Brooklyn Radiological Society
Secretary, Dr. Isadore Katz, 450 Clarkson Ave., Brooklyn 3, N. Y. Meets first Thursday of each month October through May.

Buffalo Radiological Society
Secretary, Dr. Kenneth H. Seagrave, 537 Delaware Ave., Buffalo 2, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Radiological Society
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. William B. Schwartz, 1500 W. Third Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary, Dr. William F. Hutson, 5145 N. California Ave., Chicago, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary, Dr. Norman E. Berman, 14404 S. Park Blvd., Shaker Hgts. 20, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

Colorado Radiological Society
Secretary, Dr. Bertram L. Pear, 3705 East Colfax Ave., Denver 6, Colo. Meets third Friday of each month at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. Paul J. Kingston, 114 Woodland St., Hartford, Conn. Meets first Friday in February and April.

Dallas-Fort Worth Radiological Club
Secretary, Dr. W. H. Neil, 1217 W. Cannon St., Fort Worth, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

East Tennessee Radiological Society
Secretary, Dr. J. Marsh Frere, Jr., 205 Medical Arts Building, Knoxville, Tenn. Meets in January and September.

Eastern Radiological Society
Secretary, Dr. John D. Osmond, Jr., Euclid-Glenville Hospital, Cleveland 19, Ohio. Annual Meeting to be announced.

Florida Radiological Society
Secretary, Dr. John P. Ferrell, 166 4th Ave., St. Petersburg, Fla. Meets twice annually, in the spring with the annual State Society Meeting, and in the fall.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Joseph C. Rush, 1501 Jungle, St. Petersburg, Fla.

Georgia Radiological Society
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. Donald H. Altman, 2751 Coral Way, Miami, Fla. Meets monthly third Wednesday at 8 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Radiological Society
Secretary, Dr. Harvey A. Humphrey, 462 N. Taylor, St. Louis 8, Mo.

Houston Radiological Society
Secretary, Dr. Edward B. Singleton, 6621 Fannin St., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. Claude W. Barrick, St. Alphonsus Hospital, Boise, Idaho. Meets in spring and fall.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in spring and fall.

Indiana Roentgen Society
Secretary, Dr. David E. Wheeler, 1500 North Ritter, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Lewis G. Allen, 807 Huron Bldg., Kansas City, Kansas. Meets in spring with State Medical Society, and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Robert H. Akers, V. A. Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. C. P. Naidorf, 411 Parkside Ave. Brooklyn 26, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Denis C. Adler, 2010 Wilshire Blvd., Los Angeles 57, Calif. Meets second Wednesday of month in September, November, March, April and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Edward C. Porter, 489 State Street, Bangor, Maine. Meets in June, September, December and April.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. Hollis H. Halford, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. S. F. Johnson, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. Joseph F. Wepfer, 5000 W. Chambers St., Milwaukee 10, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Bernard T. Hickman, University Medical Center, Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. J. K. Boughn, 35 11th Ave., Helena, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Alan E. Baum, 100 Nowbridge Rd., Hicksville, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. Ronald E. Waggener, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Nebraska. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. John E. Gary, 1180 Beacon St., Brookline 46, Mass. Meets third Friday of each month, October through May, at The Longwood Towers, Brookline, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Albert A. Dunn, 622 W. 168th St., New York, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. Owen Doyle, 906 Dover Road, Greensboro, North Carolina. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. R. F. Raasch, Post Office Box 990, Dickinson, North Dakota. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Paul A. Mori, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Irving Van Woert, Jr., Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. Paul D. Meyer, 125 S. Grant Ave., Columbus, Ohio. Annual meeting to be announced.

Oklahoma State Radiological Society
Secretary, Dr. J. Murphree, 617 E. Brookfield, Ponca City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Barbara J. Radmore, 1162 Willamette, Eugene, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. J. Arch Colbrunn, 19 Royal Oak Drive, Roseburg, Ore. Annual meeting to be announced.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Walter P. Bitner, 234 State St., Harrisburg, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Robert B. Funch, Department of Radiology, Germantown Hospital, Philadelphia 44, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Charles N. Chasler, 3500 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenley Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. Stanley J. Lucas, 417 Provident Bank, Cincinnati 2, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Louis L. Buzaid, The Queen's Hospital, Box 861, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. Gerhard W. Schottman, 4400 J. C. Nichols Pkwy., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Robyn Hardy, 4324 Magnolia St., New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Austin J. Tidaback, 912 Prospect Ave., Plainfield, N. J. Meets at Atlantic City at time of State Medical Society meeting and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr Mario C. Gian, 610 Niagara St., Buffalo 1, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Joseph F. Linsman, 436 N. Roxbury Dr., Beverly Hills, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Robert H. Greenlaw, 188 Irvington Rd., Rochester 20, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 11–13, 1960.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Ralph J. Littwin, Bristol Hospital, Bristol, Conn. Meetings are held bi-monthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. William E. Sheely, 1835 Eye St., N.W., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association.
Secretary. Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting to be announced.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. Richard G. Lester, 412 Union St., S.E., Minneapolis 14, Minn. Annual meeting: Ambassador Hotel, Atlantic City, N. J., Sept. 26, 1960.

Society of Nuclear Medicine
Secretary, Dr. Robert E. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. Wayne Reeser, 804 Burroughs St., Conway, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Ralph S. Clayton, 1501 Arizona, Bldg. 2-A, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. James J. Range, P.O. Box 324, Johnson City, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Texas. Next meeting January 20 and 21, 1961, Texas Hotel, Fort Worth, Texas.

Tri-State Radiological Society
Secretary, Dr. James R. Mathews, 118 S. E. First St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Richard Y. Card, St. Mark's Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Frank A. Kearney, II, 110 S. Curry St., Phoebus, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Joseph T. Houk, 14303 Ambaum Blvd., Seattle 66, Wash. Meets third Monday of each month from September through April at the University of Washington Medical School.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, 112 N. Woods St., Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Richard P. Avondu, Yonkers General Hospital, Park & Ashburton Ave., Yonkers, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual meeting each spring in various places.

X-Ray Study Club of San Francisco
Secretary, Dr. John H. Heald, 450 Sutter St., San Francisco 8, Calif. Meets monthly, third Thursday at 7:30 P.M., Children's Hospital, September through June.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Julio Toriello, 11 Calle 2-37, zona 1, Guatemala. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vega Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. Guillermo Santín. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que. Meets four times a year.

British Institute of Radiology Incorporated with the Röntgen Society
Honorary Secretary, Dr. John Blewett, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. Jean-Louis Léger, 1555 Summerhill Ave., Montreal 25, Que. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. F. McConnell, 1650 Cedar Ave., Montreal, Quebec. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Louis Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

O Colégio Brasileiro de Radiologia
Secretary-General, Dr. Walter Bomfins Pontes. Seat: Rua Major Quedinho, 99-2°. Andar, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Juan Sabbagh Dacla, Merced 565, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25-31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.]

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut. Lagenbeckstr. 1, Mainz, Germany. Annual meeting to be announced.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The next list of Meetings of Radiological Societies will be published in the September issue of the Journal.

DR. W. WALTER WASSON HONORED

W. Walter Wasson, M.D., Denver radiologist received the Norlin Award—highest decoration given by the Associated Alumni of the University of Colorado—at the June 2, 1960, University commencement exercises in Boulder. The Norlin Award was named for the late President George Norlin of the University of Colorado and is given annually to a person or persons who attended the University and later attained great distinction in their fields of endeavor.

Dr. Wasson was cited for being a "distinguished physician and eminent pioneer in the field of radiology—who has brought great honor to his profession, his State, and his University."

"In his fifty years of intense application certain significant achievements have occasioned this award." Among these are an investigation of the first aeration of the lungs of the newborn in the delivery room at St. Anthony's Hospital; the study of respiratory diseases, sinus disease, the anatomy of the lungs and the physiodynamics of respiration followed in sequence; and a report on the intrapulmonary air pressure and its relation to pulmonary capillary flow, leading to the publication in 1954 of the book, "The Auxiliary Heart."

SYMPOSIUM SPONSORED BY ORNL AND ORINS

A symposium designed to provide an introduction to thermonuclear plasma physics will be held in Gatlinburg, Tennessee, August 22–26, 1960.

Further information concerning this symposium is available from the University Relations Division, Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee.

QUEENS HOSPITAL CENTER ANNOUNCES COURSES

The following courses are announced by the Queens Hospital Center of the Department of Hospitals of the City of New York:

(1) Course in Operative Radium Therapy to be held on Wednesday mornings (9 A.M. to 12 Noon) from October 5, 1960 to November 30, 1960. Tuition: $100.00.

(2) Course in Special Radiation Procedures in the Treatment of Cancer, consisting of specialized radiation techniques. This course will commence October 17, 1960, and will last one week. Tuition: $150.00.

(3) Course in Radioactive Isotopes for Technicians, consisting of 8 sessions of four hours each, to be held every Wednesday afternoon, commencing September 28, 1960 (1 to 5 P.M.). Tuition: $100.00.

For further information please write to Dr. Philip Kahan, Supervising Medical Superintendent, Queens Hospital Center, 82–68 164th Street, Jamaica 32, New York.

It is with profound regret that we announce the death on May 27, 1960 at Santa Barbara, California of Dr. James T. Case, distinguished member and past President of the American Roentgen Ray Society and the American Radium Society.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Intracranial Calcification. By Fermo Mascherpa, Chief of the X-Ray Department, Milan Neurological Institute, Milan, Italy; and Vincenzo Valentino, Radiologist of the Institute of Semeiological Medicine, Naples University; Neuroradiologist of the Hospital "A Cardarelli," Naples, Italy. Cloth. Price, $9.50. Pp. 150, with 98 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Ave., Springfield, Ill., 1959.

Acting on the principle that a picture is worth a thousand words, the authors have assembled a splendid monograph on a subject that has long been fascinating to all radiologists.

This is a compact book with illustrations on virtually every page of a large collection of personal cases. The text is brief, concise and beautifully organized in outline form—a delight to read in a few sittings, yet having the value of a reference text.

Nonpathologic and pathologic forms of calcification are discussed with detailed information on the manner in which calcium is deposited selectively and differentially, not only in tumors but in parasitic, inflammatory, granulomatous, vascular, degenerative and endocrine diseases.

Radiologic literature is replete with divers articles on this subject presenting information which the authors have selectively condensed into their book. True, very little is new, but the material has been assembled in fine fashion and represents, in word and picture, a much needed monograph.

In the foreword Doctor Donald L. McRae succinctly expresses our opinion of this monograph by stating, "The text is concise, yet the authors have expressed their opinions and criticisms freely. I think Doctors Mascherpa and Valentino have performed a real service for neurologists, neurosurgeons and radiologists by collecting this material."

W. George Belanger, M.D.

Neurological Complications of Lymphomas and Leukemias. Publication Number 357 in the American Lecture Series; A Monograph in the Bannerstone Division of American Lectures in Tumors. By Henry M. Williams, M.D.; Henry D. Diamond, M.D., F.A.C.P.; Lloyd F. Craver, M.D., F.A.C.P.; and Herbert Parsons, M.D. Edited by David A. Karnofsky, M.D., Associate Professor of Medicine, Cornell University Medical College, New York, N. Y. Cloth. Price $5.75. Pp. 134. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1959.

This comprehensive review of the neurologic complications in lymphomas and leukemias deals with 795 such findings in 745 patients. Spinal cord compression, cerebral manifestations, cranial and peripheral nerve involvement, herpes zoster, pituitary involvement, and central nervous system infection and hemorrhage constitute the main topics of discussion. The relative incidence of each complication, its pathogenesis, clinical signs and management are presented and comparison is made with reports in the literature.

The material in each chapter is clearly organized and tabulated and the conclusions drawn are in strict keeping with the factual data. There is much useful information in this small volume for the physician who is concerned with the management of patients with leukemias and lymphomas.

William S. Reveno, M.D.

Preventive Medicine; Principles of Prevention in the Occurrence and Progression of Disease. Edited by Herman E. Hilleboe, M.D., Commissioner of Health, State of New York, Department of Health, Albany, New York; and Granville W. Larimore, M.D., Deputy Commissioner of Health, State of New York, Department of Health, Albany, New York. Cloth. Price, $12.00. Pp. 731, with 59 illustrations. W. B. Saunders Company, West Washington Square, Philadelphia 5, Pa., 1959.

The thirty-one contributors to this book set forth a new way to look at preventive medicine for medical students, general practitioners, specialists and professional workers in official and voluntary health agencies.

The first of the three sections into which the book is divided deals with disease prevention, prophylactic measures, nutrition, the elimination of conditions which predispose to disease, and preventive dentistry.

In the second section, the prevention of the progression of disease is discussed at length from the viewpoint of periodic health inventories, early detection, follow-up of screening and diagnostic examinations, rehabilitation, alcoholism, and narcotic addiction.

Finally, in the third section the role of education in preventive medicine is analyzed and the services which aid in the practice of preventive medicine are described.

The scope of the book is broad; it includes consideration of ionizing radiation, as it applies to the field of preventive medicine, as well as treatment of the diseases which are discussed. There is a complete index; the type is large and easy to read and the text is adequately documented. This book can be highly recommended since it represents the latest knowledge in this important specialty.

Robert C. Moehlig, M.D.

BOOKS RECEIVED

Collected Papers from The Radiumhemmet and The Radiopathology Institute of King Gustaf V's Jubilee Clinic. Volume VIII: 2, 1957–1958. Cloth. Pp. 66, with numerous illustrations. Karolinska Sjukhuset, Stockholm, Sweden, 1959.

Acta Anatomica. Vol. 38—Supplementum 36. Spinal Joint Changes and Dental Infections. By Bo E. Ingelmark, Vilh. Møller-Christensen, and Ove Brinch. Paper. Pp. 60, with 27 illustrations. S. Karger AG., Arnold Böcklinstrasse 25, Basel, Switzerland, 1959.

Selected Papers from the Institute of Cancer Research: Royal Cancer Hospital and from the Royal Marsden Hospital, 1957. Volume 12. Cloth. Pp. 1099. Institute of Cancer Research, Royal Cancer Hospital, The Chester Beatty Research Institute, Fulham Road, London, S.W.3, England.

Nefrocalcinosis: Estudio Clinico-Radiológico. (Thesis). By Dr. Manuel Viamonte Puncet. Paper. Pp. 95. Molina y Compania, S. A., Sol Num. 316, Habana, Cuba, 1959.

Low-Level Irradiation. Edited by Austin M. Brues. Cloth. Price, $3.75. Pp. 148. American Association for the Advancement of Science, 1515 Massachusetts Ave., N. W., Washington 5, D. C., 1959.

Radiologic Records. By Sister Christina Spirko, C.SJ., R.T., B.S. in R.T., M.A., F.A.S.X.T., Associated with the Department of Roentgenology, St. Mary's Hospital, Amsterdam, New York; Instructor in Sociology, St. Mary's School of Nursing, Amsterdam, New York; Editor, X-Ray Column, Hospital Management, Chicago, Illinois. Cloth. Price, $8.50. Pp. 304. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1960.

X-Ray Technology. Second edition. By Charles A. Jacobi, B.Sc., R.T.(A.R.X.T.), M.T.(A.S.C.P.), Chairman, Medical X-Ray Technology, Oregon Technical Institute, Oretech Branch, Klamath Falls, Oregon; Chairman, Education Committee, Oregon Society of X-Ray Technicians; Chairman, Education Committee, Northwest Conference of X-Ray Technicians; formerly, Chief X-Ray and Medical Technologist, Medical Services Division, Atomic Energy Commission, National Reactor Testing Station, Idaho Falls, Idaho; and Donald E. Hagen, R.T.(A.R.X.T.), Technical Supervisor for C. Todd Jessell, M.D., and George R. Satterwhite, M.D., Radiologists, Portland, Oregon; formerly, Instructor, Medical X-Ray Technology, Oregon Technical Institute, Oretech Branch, Klamath Falls, Oregon. Cloth. Price, $10.00. Pp. 452, with 320 illustrations. C. V. Mosby Co., 3207 Washington Blvd., St. Louis 3, Mo., 1960.

Klinische Physiologie; aktuelle Probleme in Übersichten. By Dr. W. A. Müller, Chefarzt der II. Inneren Abteilung des Robert-Bosch-Krankenhauses, Stuttgart. Paper. Price, $5.50. Pp. 118. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1960. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Harmful Effects of Ionising Radiations. By Ethel Browning, M.D., Formerly, H.M. Medical Inspector of Factories, Ministry of Labour and National Service, London. Paper. Price, $3.00. Pp. 158. D. Van Nostrand Company, Inc., 120 Alexander St., Princeton, N. J., 1960.

Anatomy; A Regional Study of Human Structure. By Ernest Gardner, M.D., Wayne State University; Donald J. Gray, Ph.D., Stanford University; and Ronan O'Rahilly, M.Sc., M.D., Wayne State University. Cloth. Pp. 999, with numerous illustrations. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, Pa., 1960.

Acta Orthopaedica Scandinavica. Supplementum No. 41. Fractures of the Distal End of the Radius; a Clinical and Statistical Study of End Results. By Anders Lidström. Paper. Pp. 118, with illustrations and tables. Ejnar Munksgaard, Copenhagen, Denmark, 1959.

Vol. 84, No. 1

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Mones, Robert, and Werman, Robert. Pantopaque myeloencephalography. *Radiology*, June, 1959, *72*, 803–809. (Address: R. Mones, The Mount Sinai Hospital, Fifth Ave. and 100th St., New York 29, N. Y.)

A new procedure is described for the demonstration of the posterior fossa and upper cervical region. By this technique it is possible to fill the third and fourth ventricles, cisterna magna, and posterior rim of the foramen magnum with pantopaque instilled by way of a lumbar puncture.

Nine to twelve cc. of pantopaque is injected into the lumbar subarachnoid space, as for routine myelography. The spinal needle is then removed; the patient is turned on his back and then, under fluoroscopic control, is tilted cephalad 45 to 55 degrees. The fourth ventricle, cisterna magna, and posterior aspect of the third ventricle usually fill within a few minutes. Anteroposterior and lateral roentgenograms are taken and then the patient is brought to a horizontal position, where another series of exposures is made. If the films are satisfactory, the patient is placed with the head in the erect position for five minutes. Pantopaque drains out of the ventricles and cisterns, pools in the lumbar region and is then removed.

This procedure, which is designated as myeloencephalography, was performed in 40 patients. One patient had a reaction characterized by fever, pleocytosis and meningeal signs. No other reactions or sequelae were seen. Headache or a sense of fullness in the head were common complaints but the discomfort was less than that experienced during pneumoencephalography. In every instance it was possible to define the posterior rim of the foramen magnum. Filling of the fourth ventricle was obtained in 23 cases, with nonfilling in 17 cases. Among the 17 cases without ventricular filling were 11 associated with posterior fossa masses. Brain stem disease was diagnosed by this method in 29 cases.

In certain cases important information which is not obtainable by pneumoencephalography is obtained by myeloencephalography. It is particularly valuable for studying structures of the posterior fossa. Burr hole ventriculography can frequently be avoided. The procedure is also useful in diagnosing abnormalities of the posterior foramen magnum, such as herniated cerebellar tonsils, bony abnormalities, Arnold-Chiari malformations, and tumors.—*Arno W. Sommer, M.D.*

Wilson, McClure, and Snodgrass, S. R. Positive contrast ventriculography. *Radiology*, June, 1959, *72*, 810–815. (Address: McC. Wilson, University of Texas Medical Branch, Galveston, Texas.)

Complete and unequivocal roentgenographic visualization of the third ventricle, the aqueduct of Sylvius and the fourth ventricle is sometimes imperative during the course of a neuroroentgenologic investigation of the brain. This can usually be obtained with the use of air as the contrast medium, but at times adequate visualization is not evident even when laminagraphy is resorted to. It is in these situations that a little used technique, called positive contrast ventriculography, utilizing a high density contrast material such as pantopaque may be of great value.

During the year 1957, 243 air contrast studies of the brain were done. Positive contrast ventriculography was performed in 11 of this number. No false positive findings were encountered and complications were infrequent. Infants who had massive dilatation of the ventricular system and in whom pneumoventriculography had been performed earlier in the day had, at times, a high fever for a day or so following the procedure. The contrast medium, usually 1 to 2 cc., is introduced by means of a needle through a surgical burr hole in the lateral ventricle. The technique used by the authors is described in detail and 4 cases are presented along with photoroentgenograms, illustrating the value of this procedure. The question of late effects of pantopaque on the ependyma is discussed, and the authors believe that, in selected cases, the advantage of this procedure as a diagnostic tool outweighs the possible disadvantage of late adverse effects on the ependyma.

Positive contrast ventriculography is of greatest value in demonstrating lesions in the region of the midbrain and pons, particularly in the study of infants with noncommunicating hydrocephalus. This procedure is also of definite value in some cases of lesions around the third ventricle and in the cerebellum. Unequivocal localization of neoplasms involving the pons or mesencephalon has helped to avoid unnecessary negative operations and has established the need for other therapeutic measures.—*Donald N. Dysart, M.D.*

McDowell, Fletcher H., Schick, Robert W., Frederick, William, and Dunbar, Howard S. An arteriographic study of cerebrovascular disease. *A.M.A. Arch. Neurol.*, Oct., 1959, *1*, 435–442. (From: Cornell Neurological and Neurosurgical Service, Bellevue Hospital, New York, N. Y.)

Cerebral angiographic studies were performed on 68 patients with 69 strokes. Seventy per cent of the patients in this series with severe neurologic defects were found to have an occlusion in a major cerebral artery. The artery most frequently occluded was the middle cerebral (18 of 68 patients, or 26.4 per cent). The anterior cerebral artery was found to be occluded distal to the junction with the anterior communicating artery only once in this series. An occlu-

sion of the anterior cerebral artery between its origin and the anterior communicating artery could easily occur without symptoms, as this artery fills adequately from both internal carotid arteries.

Partial occlusion of the carotid or vertebral artery circuit was observed four times. Vertebrobasilar occlusion was found only once. Three patients were found by arteriography to have an intracerebral hematoma. There was no instance in the series in which arteriography was helpful in distinguishing a cerebral thrombosis from an intracerebral hemorrhage prior to anticoagulant therapy, when the bedside examination did not strongly suggest one in favor of the other.

Three patients of this series were found to have nonvascular lesions. Two had subdural hematoma and 1 had a tumor. Twenty patients showed arterial abnormalities, such as tortuosity of the larger arteries, small irregular defects in the arterial wall, and irregular dilatation or constriction of the internal carotid artery or its branches. These are commonly considered as evidence of atherosclerosis. "Short filling," or the inability of the smaller arteries to fill, or delay in filling, was noted frequently and cannot be adequately explained.

In general, patients with internal carotid artery occlusion had less severe clinical neurologic defects than those with middle cerebral artery occlusion. Twenty of the patients with moderate or severe clinical defects failed to show any correlation because of the absence of demonstrable occlusions. Since their arteriograms were not normal, this study would indicate nonocclusive degenerative cerebrovascular disease of sufficient degree to account for the clinical findings seen in patients without major occlusions.

The authors conclude that angiography is not advisable or helpful in the diagnosis of the usual patient with an adequate history and clinical findings suggesting stroke. With an inadequate or no history arteriography may aid in eliminating the possibilities of subdural hematoma, intracerebral hematoma and brain tumor. Carotid arteriography should not be performed in patients with cerebrovascular disease if they are in coma or stupor or if their clinical state is steadily worsening.—*J. N. Ané, M.D.*

Pyo, John, and Lowman, Robert M. The "ponticulus posticus" of the first cervical vertebra. *Radiology*, June, 1959, *72*, 850–854. (Address: Robert M. Lowman, Department of Radiology, Yale Medical Center, New Haven, Conn.)

The ponticulus posticus is an anatomic variation of the first cervical vertebra which consists of a bony arch across the vertebral artery sulcus. This arch is the result of an anomalous ossification center in the posterior atlanto-occipital ligament. The usual opening in the ligament, for the passage of the vertebral artery, is thereby converted into a bony aperture. Although some authors state that the ponticulus posticus has no clinical significance, others report that enlargement of the grooves and foramina of the first cervical vertebra indicates vertebral artery enlargement. To further evaluate these reports, the authors have studied the ponticulus posticus with respect to incidence, appearance, and dimensions.

The skull roentgenograms of 300 normal persons were reviewed. These consisted of 170 males and 130 females ranging in age from newborn to ninety years. A ponticulus was demonstrated in 38 cases (12.6 per cent). This finding was bilateral in 26 and unilateral in 12 cases. Most of these occurred in the age group beyond forty and the sex incidence was approximately equal. There were numerous variations in respect to size, position and configuration and the bony bridging varied in completeness and degree of ossification. Where the ossification was incomplete, the defect usually was located at the posterior margin, although it could be found at any portion of the arch. Some cases showed thinning of the posterior arch of the atlas along the vertebral artery sulcus. To determine the normal variation in size of the ponticuli postici, the measurements of each were made directly on the film. This revealed an average size of 8.5 mm. with a range of 6 to 11 mm. in females and 6 to 12 mm. in males.

The results of this study indicate that there is a wide variation in the size, shape and contour of the ponticulus posticus and that complete or partial bony bridging occurs in 12 to 13 per cent of the population, principally in the age group beyond forty. The authors conclude that minimal or moderate changes produced by an enlarged vertebral artery may be difficult to recognize.—*Edward B. Best, M.D.*

Neck and Chest

Nivière, J., Larcan, A., and Simonel, A. Les aspects radiocliniques et étiologiques des pneumopathies aiguës virales (à propos de 175 observations). (Radioclinical and etiologic aspects of acute viral pneumopathies [based on 175 cases].) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1959, *40*, 519–528. (From: Services d'Electroradiologie et de Pneumophtisiologie de l'Hôpital militaire "Sédillot," Nancy, France.)

Atypical pneumophaties include all acute nontuberculous infectious pneumopathies except classic pneumonia. Systematic mass roentgen-ray examinations and the development of direct and indirect viral techniques have proved that these pneumopathies are frequent. The development of viral pathology is the most remarkable aspect of present day infectious pathology.

After reviewing the clinical aspects of acute viral pneumopathies, the authors analyze the roentgen-

ologic signs which they classify in two categories:

(1) *Typical Signs.* (a) The basal lesions present a variable picture. They may appear as a vague, faint shadow superimposed on an accentuated bronchovascular triangle, which is also known as the hilifugal pneumonitis of Glanzmann and may extend into the costophrenic sulcus. Other forms are the nodular infiltration which is not always homogeneous, is generally rather hazy, and only occasionally well delineated; and the small dense nodules (of bronchoalveolitis) in one or both bases which clear up rather rapidly to be replaced by a reticular aspect of the parenchyma, probably due to peribronchovascular infiltration. The lateral roentgenogram does not show any segmental distribution. These lesions are predominantly localized in the right base. When the left base is involved in its medial portion, blurring of the left cardiac border and blunting of the left costophrenic angle are apparent. (b) The segmental lesions may involve a lobe or a segment. Their distribution is best shown on lateral roentgenograms. The bronchus is frequently the site of involvement.

(2) *Atypical Signs.* These are relatively evanescent and may imitate more serious diseases. They are rarer than the preceding signs. (a) False hilar shadows, attributable to involvement of the inner part of the apex of the lower lobe, are easily detected on the lateral roentgenogram. The lymph node enlargement is usually small and accompanies an infiltrative process that may partially cover a segment. Middle parahilar involvement might suggest a primary complex and even carcinoma. The diagnosis rests upon clinical and biologic investigation. (b) Heterogenous infiltrations, when located in the cleidohilar region, resemble tuberculosis, sometimes even with cavitation, as seen on laminagrams. This is due probably to central resorption of the infiltrate or to bronchial involvement with ventilatory disturbance. (c) Accompanying lesions show pleural and/or pericardial effusion. (d) The miliary form is rarely generalized, sparing the apices and bases, and it resolves rapidly. (e) The atelectatic form is located in the bases, close to the diaphragm. It may appear plate-like (Fleischner's line) or segmentary due to involvement of a proximal bronchus.—*H. P. Lévesque, M.D.*

McKay, Eric, and Richardson, Janet. Pneumocystis Carinii pneumonia associated with hypogammaglobulinaemia. *Lancet,* Oct. 31, 1959, *2,* 713–715. (From: Royal Aberdeen Hospital for Sick Children, Aberdeen, Scotland.)

This pulmonary affliction, a form of interstitial pneumonia, is a fairly common disease in infancy on the continent of Europe. It afflicts weak and immature babies. Abnormalities of the plasma proteins are not generally described; recent British reports, however, point out the apparently frequent coexistence of low gamma globulin levels.

The authors cite a case report describing a nine month old male; two of his siblings suffered congenital hypogammaglobulinemia, and one of them died from pneumocystis Carinii. Three weeks prior to admission, listlessness, irritability and dry cough were noted. He became increasingly tired and breathlessness on exertion was evident, with slight cyanosis of the nail beds. The examination showed early clubbing of the fingers and toes. The pulse rate was 156 and the respiratory rate 48. The temperature was normal. The ausculation of the chest showed diminished breath sounds over the mid-right lung but no adventitious sounds were heard and the percussion note was unimpaired. No other physical findings were present.

A roentgenogram of the chest showed extensive opacities in both lungs with partial collapse of the right lower lobe.

Electrophoretic evaluation of serum proteins revealed reduction in gamma globulin. Cultures of throat and cough secretions were sterile or, on occasion, showed *Streptococcus viridans.*

He was treated with gamma globulin and various antibiotics plus prednisolone. His progress was steadily downhill. Oxygen therapy was necessary. It was required continuously and in increasing concentrations for two and one-half months until death.

The major autopsy findings were in the lungs, which were firm and fleshy and fairly uniformly solid. They were pink with scattered whitish areas and were unusually dry. There was no distortion of the bronchial tree and no evidence of pus. There was no pleural reaction. A culture produced a scanty growth of *Staphylococcus aureus.*

Histologic examination showed evidence of an interstitial pneumonia, the alveolar septa being thickened and infiltrated by lymphocytes and large mononuclear cells. Plasma cells were conspicuous by their absence. In some areas, the alveoli and alveolar ducts were distended and contained an exudate which was positive to periodic acid-Schiff staining. The histologic findings were thought to be pathognomonic of pneumocystis pneumonia.

This entity should be suspected in the patient who exhibits signs of oxygen starvation, failure to respond to antibiotics, and a paucity of auscultatory signs. In this disease the roentgenologic changes are out of proportion of the clinical signs and extensive infiltrations may be noted on the chest roentgenogram. The etiologic agent is not known but this case supports the association between this disease and hypogammaglobulinemia.—*Jack Reynolds, M.D.*

Jahn, O. Die Resorption von Joduron B bei chronischer Pneumonie, deformierender Bronchitis und Bronchiektasie. (The absorption of joduron B in chronic pneumonia, deforming bronchitis and bronchiectasis.)

Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin, Oct., 1959, *91*, 478–482. (From: Medizinische Universitätsklinik [Lung genstation Natters], Innsbruck, Austria.)

Absorption of contrast material from the lungs after bronchography depends on the condition of the bronchial mucosa. The greater the pathologic changes in the bronchi the longer the contrast material will remain.

Laminagraphic studies following bronchography with joduron B showed that the contrast material is visible for twenty minutes in normal lungs; forty minutes in chronic pneumonia; fifty-five minutes in deforming bronchitis; and eighty minutes in bronchiectasis. It was also noted that the absorption rate in a neoplasm is the same as in normal tissue.—*J. Zausner, M.D.*

Nelson, Sidney W., Christoforidis, Anthimos, and Pratt, Phillip C. Barium sulfate and bismuth subcarbonate suspensions as bronchographic contrast media. *Radiology*, June, 1959, *72*, 829–838. (Address: S. W. Nelson, Ohio State University Health Center, Columbus, Ohio.)

The authors report the results of an investigation of the use of the barium sulfate and bismuth subcarbonate suspensions in the tracheobronchial tree, first in animals and subsequently in man.

Four groups of rabbits were used for parallel studies with dionosil oily, lipiodol, bismuth subcarbonate and barium sulfate. The barium sulfate suspension consisted of one part by weight of micropaque and two parts by weight of a solution of normal saline containing 1.5 per cent sodium carboxymethylcellulose. Animals from each of the four groups were sacrificed at intervals of twenty-four hours, one week and ten weeks following the administration of the contrast medium transtracheally with a hypodermic needle. Chest roentgenograms were obtained after each injection to confirm the filling of the lower lobe bronchi and to follow elimination of the contrast material. No significant histopathologic differences in the acute effects of the four contrast media were demonstrated. The histopathologic response was predominantly a foreign body reaction. The most intense and prolonged reaction was that seen in the lungs following the use of bismuth subcarbonate. It was therefore decided to abandon the use of the bismuth preparation. No permanent histopathologic effects of dionosil oily, lipiodol or barium sulfate were found at ten weeks.

Clinical investigations of the use of barium sulfate as a bronchographic contrast medium were carried out after animal experiments demonstrated its safety. Fifty patients in whom barium sulfate was used were compared with a parallel group of 50 patients receiving dionosil oily. In every patient the temperature and pulse were recorded after bronchography. Twenty-three of 50 patients had a pulse rate above 100 per minute within twenty-four hours following dionosil bronchography, whereas only 11 out of 50 showed a similar pulse rate elevation following the use of barium sulfate. No patient exhibited a temperature elevation of more than twenty-four hours' duration following the use of barium sulfate, whereas 4 of the 50 who had received dionosil still showed temperatures above normal beyond that period.

Roentgenographic contrast and coating of the bronchial mucosa were excellent with barium sulfate. All types of pathology were easily demonstrated. In 47 out of 50 patients there was complete disappearance of the barium within seven days as judged by postbronchography chest roentgenograms. There was no clinical or experimental evidence of chronic fibrosis or foreign body granuloma resulting from the use of barium or sodium carboxymethylcellulose.—*Arno W. Sommer, M.D.*

Johnson, Philip M., and Irwin, George L. An evaluation of the pharmacological hazards resulting from use of "visciodol" in bronchography. *Radiology*, June, 1959, *72*, 816–828. (Address: P. M. Johnson, The School of Medicine, The University of North Carolina, Chapel Hill, N. C.)

The purpose of this paper is to report a study of sulfanilamide absorption and methemoglobin formation after bronchography with visciodol, a suspension of finely powdered sulfanilamide in lipiodol, the sole function of the sulfanilamide being to increase the viscosity of the lipiodol.

Spectrophotometric determinations of blood sulfanilamide and methemoglobin levels were made in 35 patients prior to bronchography and at intervals thereafter. Alveolar spillage and the volume of visciodol ingested were quantitatively estimated. Following administration of visciodol, sulfanilamidemia invariably occurred, and the mean concentration of sulfanilamide in the serum exceeded the accepted therapeutic range between the second and sixth hours after bronchography. In general, the higher blood sulfanilamide levels occurred with use of larger volumes of visciodol. Methemoglobin formation occurred in 29 patients, and the highest levels were with the largest doses of visciodol. Overt cyanosis was observed at the higher levels. Both alveolar spillage and visciodol ingestion enhanced blood sulfanilamide and methemoglobin values. Methylene blue proved effective in reducing blood methemoglobin levels. Animal studies indicated a greater absorption of sulfanilamide from the lungs than from the gastrointestinal tract.

The occurrence of sulfanilamidemia and methemoglobinemia following visciodol bronchography carries a small but unjustifiable risk. Certain precautions

which should be taken when the procedure is employed are suggested.—*Walter H. Jarvis, Jr., M.D.*

Jacobson, Harold G., Rubinstein, Berta M., and Escher, Doris J. W. Opacification of an atelectatic lung segment during selective angiocardiography. *Radiology*, July, 1959, *73*, 95–99. (Address: H. G. Jacobson, Montefiore Hospital, 210th St. and Bainbridge Ave., New York 67, N. Y.)

There has been no report in the recorded literature thus far of the demonstration of the vasculature in an atelectatic lung segment in man using selective angiocardiography. Various investigators have demonstrated in animal experiments that atelectatic lung has a diminished blood supply. Studies of the blood oxygen saturation curves in such experiments, however, have shown that there is no change in the oxygen saturation of the arterial blood even with the total collapse of one lung. Björk, working with dogs, reported in 1953 that in acute atelectasis of one lung, especially in the first few hours, there is little or no significant decrease in blood flow through the atelectatic segment. After the atelectasis had persisted for one and one-half months, practically no opacified blood could be seen passing through the collapsed segment. Bulgarelli, using angiocardiographic techniques, in 1955 reported hypovascularity of the atelectatic lung with hypercirculation and vascular dilatation in the opposite lung in a man with left lower lobe atelectasis.

The present report is of a six year old white boy who developed acute right upper lobe atelectasis following anesthesia in preparation for angiocardiographic studies for a suspected interventricular septal defect. A series of roentgenograms shows the vascular opacification of the atelectatic lung segment. As expected, the vasculature in this segment showed significant compression. Retention of opaque medium was evident long after the normal adjacent lung had cleared.

Although these lung findings were incidental to the intended examination, the authors further the idea that the techniques of angiocardiography may be utilized in studying suspected or known pathology in the lungs.—*Z. Petrany, M.D.*

Bergonzini, R., and Calzavara, M. Contributo casistico e rassegna della letteratura sui tumori maligni primitivi del cuore. (Case report and review of the literature on primary malignant tumors of the heart.) *Radiol. med.*, Sept., 1959, *45*, 865–886. (From: Istituto di Radiologia dell'Università di Modena, Modena, Italy.)

The authors describe a case of primary cardiac malignancy which is classified as reticulosarcoma (histiocytoblastoma) of the right ventricle associated with pulmonary and hepatic metastases. The diagnosis in this thirty-two year old patient was not made before autopsy, principally because of the rapidity of the course of the disease.

The authors discuss the various diagnostic problems in their study of 182 cases. They stress the possibility of an exact *intra vitam* diagnosis by means of angiocardiography and of surgical therapy.—*Anthony A. Blasi, M.D.*

Singleton, Edward B., McNamara, Dan G., and Cooley, Denton A. Radiologic features of isolated ventricular septal defects. *A.M.A. Arch. Surg.*, Nov., 1959, *79*, 715–723. (From: Department of Radiology and the Cardiac Clinic, Texas Children's Hospital, and the Cora and Webb Mading Department of Surgery, Baylor University College of Medicine, Houston, Texas.)

The roentgenologic features of isolated ventricular septal defects depend upon the size of the defect and the amount of pulmonary resistance. The physiologic alterations in the form of pressure changes result in observable anatomic or roentgenologic features which may be correlated with pressure in the pulmonary circulation and the volume of intracardiac shunts. The roentgenologic features involve changes in the size of the left and right ventricle and the left and right atrium and in the degree of pulmonary vascularity.

The manifestations of the defect have been classified as follows:

1. *Small Defect.* The intraventricular shunt is slight; the roentgenologic appearance is normal.

2. *Medium Defect.* The pulmonary vascularity is increased and the main pulmonary artery segment is prominent. Left ventricular enlargement is present. The majority of cases will show some degree of left atrial enlargement.

3. *Large Defect with Left to Right Shunt.* Large ventricular septal defects with moderate elevation of pulmonary arterial pressure demonstrate the features observed in Class 2 plus evidence of right ventricular enlargement and greater dilatation of the pulmonary artery segment. The over-all size of the heart is moderately enlarged. The left atrium is also dilated, to a greater degree than that seen in Class 2. The pulmonary vascular markings are greatly accentuated and extended to the periphery. Hilar pulsations may be noted with fluoroscopy.

4. *Large Defect with Equal Pressures.* In large defects when the pressures in the pulmonary and systemic circulations are approximately equal and the shunt is predominantly left to right, the heart is usually large and dilated. Both right and left ventricular enlargement is present and there is usually no left atrial enlargement. The peripheral pulmonary vascular shadows may be diminished in size but the hilar vessels appear larger. Because of the relatively

greater size of the right ventricle, there may be a decrease in the over-all size of the heart when compared to earlier roentgenograms.

5. *Large Defect with Right to Left Shunt.* There is severe pulmonary hypertension in this class. The heart is usually small without ventricular dilatation. The right ventricle is hypertrophied, as evidenced by elevation of the apex in the posteroanterior projection and increased convexity in the left anterior oblique view. The aorta appears small but the main pulmonary artery segment is extremely prominent. The hilar vascular markings are accentuated but become markedly attenuated in the mid- and peripheral lung field. The left atrium is not enlarged. The occasional case may appear essentially normal roentgenographically. The value of electrocardiographic data becomes of importance in these cases.

The roentgenographic appearance of the heart and lung fields in infants with large ventricular septal defects is usually not characteristic. The lesion may not have been present long enough to produce significant anatomic change and the thymus obscures the cardiac silhouette. Some information may be gained, however, when evidence of increased pulmonary vascularity and enlargement of the left atrium is noted.

Venous angiocardiography is said to play a minor role in evaluating ventricular septal defects. Retrograde aortography is of use particularly in differentiating ventricular septal defects from extracardiac shunts.

After surgery, changes in size and configuration and in pulmonary vascular pattern develop slowly over a period of several months. There may be an initial slight increase, then a gradual decrease in the size of the heart and in the prominence of the pulmonary vascular pattern.—*Jack Reynolds, M.D.*

Moore, Charles B., Kraus, William L., Dock, Donald S., Woodward, Edward, Jr., and Dexter, Lewis. The relationship between pulmonary arterial pressure and roentgenographic appearance in mitral stenosis. *Am. Heart J.*, Oct., 1959, *58*, 576–581.

The authors determined the "PA/Chest Ratio," as defined by Ozawa, in a group of 56 patients with mitral stenosis and correlated these data with the pulmonary arterial mean pressure. Positive correlation of an exponential character, resembling the shape of a pressure-volume curve, was obtained in this group of patients.

Enlargement of the pulmonary arterial segment of the cardiac silhouette is a common roentgenographic finding in mitral stenosis. Since the segment is essentially a passive, distensible conduit for blood, the pressure within it should be one of the main determinants of its size.

Fifty-six adult patients with mitral stenosis diagnosed by clinical and cardiac catheterization findings, and subsequently confirmed by surgery, were studied. Patients with spinal or thoracic deformities, left ventricular enlargement, aortic valvular disease, significant mitral regurgitation, previous thoracic surgery, or any condition which might alter the roentgenographic appearance of the pulmonary arterial segment were excluded. Pulmonary arterial pressures were measured through a cardiac catheter. Mean pressures were obtained by electrical integration.

A standard 7 foot posteroanterior roentgenogram of the chest of each patient, taken within three or four days of cardiac catheterization, was examined and the following measurements were made: (1) transthoracic diameter, *i.e.*, the greatest transverse diameter of the chest from pleura to pleura, in centimeters; and (2) pulmonary arterial size, *i.e.*, the distance from the midline, as judged by the thoracic spine, to the edge of the pulmonary arterial segment at its widest point, in centimeters. From these measurements the "PA/Chest Ratio" was calculated as follows:

$$\frac{\text{Pulmonary Arterial Size}}{\frac{1}{2}\ \text{Transthoracic Diameter}} \times 100$$

The measurements for the roentgenograms were also plotted against pulmonary arterial systolic pressure, pulmonary arterial diastolic pressure, pulmonary capillary pressure, pulmonary vascular resistance, and cardiac index. The correlations obtained from these plots were much poorer than those with pulmonary arterial mean pressure.

The authors warn that this method cannot be extended indiscriminately to other diagnostic groups in which enlargement of the pulmonary arterial segment may be associated with pulmonary hypertension. In a homogeneous diagnostic group, such as that of cases of mitral stenosis, in which the cardiac index is quite uniformly reduced or at low normal levels and pulmonary emphysema is not a factor, the "PA/Chest Ratio" allows a fairly accurate estimate of the pulmonary arterial pressure.—*J. N. Ané, M.D.*

Guzman, Santiago V., and West, James W. Cardiac effects of intracoronary arterial injections of various roentgenographic contrast media. *Am. Heart J.*, 1959, *58*, 597–607. (From: Department of Pharmacology, School of Medicine, University of Pennsylvania, Philadelphia, Pa.)

The authors studied the cardiac effects of intracoronary arterial injection of various contrast substances on coronary blood flow, myocardial contraction, and the electrocardiogram. The fatalities associated with clinical angiocardiography have been more commonly related to respiratory arrest immediately or shortly after the injection of a contrast substance. Other causes listed have been "allergic

sensitivity," cardiac arrest and acute heart failure.

These studies were performed on 22 normal adult dogs. Intracoronary arterial injections (0.025 to 0.25 cc./kg.) of diodrast 70 per cent, urokon 70 per cent, hypaque 50 per cent, renografin 76 per cent, and cardiografin 85 per cent were employed. The sequence of injections was varied in each experiment in order to avoid the influence of a previous injection on the effect of each radiopaque material.

The electrocardiographic changes following intracoronary injection of the different radiopaque substances consisted of flattening or inversion of a previously upright T wave, occasionally accompanied by ST depression. Diodrast, renografin and cardiografin elicited minimal to moderate ST-T wave changes as compared to hypaque and urokon. Urokon appeared to be the most toxic of the group, since it often produced ventricular tachycardia. In 6 of the 22 dogs studied during intracoronary arterial injection of 70 per cent urokon (0.25 cc./kg.) and in 2 dogs after intracoronary arterial injection of 50 per cent hypaque (0.25 cc./kg.), ventricular fibrillation developed. Comparative doses of diodrast (70 per cent), renografin (76 per cent), and cardiografin (85 per cent) did not produce this fatal outcome. All of the radioapque substances studied elicited a slight and transient increase in heart rate accompanied by a fall in blood pressure. Urokon, on many occasions, produced transient A-V heart block, which was not eliminated by bilateral vagotomy, but which disappeared after the administration of atropine.

The effects of the different contrast materials on myocardial contractility were studied in 10 dogs, each animal receiving from two to four injections of each of the contrast media (0.05 to 0.25 cc./kg.). The degree of myocardial depression was most marked and persistent with urokon (average decrease of 70 per cent of control) and least marked with diodrast (average decrease of 15 per cent of control). The depresssant effects of hypaque, renografin and cardiografin were of the same degree, averaging about 35 per cent of control. In small doses of from 0.025 to 0.05 cc./kg., all of the contrast media except urokon showed a stimulant effect following recovery from depression. This was more marked after the injection of diodrast. Recovery from the depressant effects of the various contrast substances was complete with all media except urokon.

Forty-eight observations in 5 dogs were made on the effects of intracoronary contrast injections on coronary blood flow. The results indicated that all of the contrast media increased coronary blood flow, averaging a 60 per cent increase over the control. Diodrast produced the greatest and most lasting effect.

Intracoronary arterial injections of the various contrast media caused a drop in systemic blood pressure. The degree of fall in pressure (from 10 to 80 mm. Hg systolic pressure) appeared to parallel the degree of change in the T wave.

Re-injection of each of the various opaque media studied showed a diminution in electrocardiographic changes and in cardiac depression.—*J. N. Ané, M.D.*

Swart, B. Die Breite der V. azygos als röntgendiagnostisches Kriterium pathologischer Kollateralkreisläufe. (The width of the azygos vein as a roentgenologic diagnostic criterion of pathologic collateral circulations.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1959, *91*, 415–444. (From: Universitäts-Strahleninstitut des Universitätskrankenhauses, Hamburg-Eppendorf, Germany.)

The caliber of the azygos vein at the right tracheobronchial angle gives valuable information concerning the type and degree of cardiac decompensation as well as the pathways of the collateral circulation from the portal and caval systems. In these days of increasing thoracic and thoracoabdominal interventions, much valuable preoperative knowledge can be imparted to the surgeon. Measurement of the azygos vein should be included in the study of all cardiac disorders beside the conventional examination of the size and configuration of the heart and the congestive changes in the lungs. Likewise, attention to this measurement should be given whenever esophageal varices are found or suspected.

After 2,000 examinations of 104 patients, using a variety of positions and techniques, the author concludes that the most significant measurements are made with the patient in a sitting position and during a mild expiratory phase of respiration. The greatest widths are obtained in the Trendelenburg position but this is uncomfortable or impossible for many patients, particularly cardiacs. In the sitting position, the values are independent of the age, body size or vital capacity and they are only slightly affected by respiration. The vein may be twice or three times larger in the recumbent than in the sitting position. Since the distributional spread of the values is much less when seated than when recumbent, the former values are more significant. However, in certain instances, the arithmetic difference between these two values is important. The mean value for the width of the azygos vein is $6.8+1.5$ mm. (seated) and 15.9 ± 2.2 mm. (recumbent).

The best means of studying the azygos vein is by laminagraphy with cuts at 9, 10, 11 and 12 cm. levels at the tracheal bifurcation. The appearance of the azygos vein is drop-like in 20 per cent, semicircular in 63 per cent and spindle-shaped in 5 per cent. The remaining 12 per cent are atypical, therefore difficult to evaluate, and represent a defect in the method. The measurement is made from the most lateral margin of the azygos vein contour perpendicular to the tracheobronchial wall. To obtain the actual size, all values must be multiplied by a factor of 0.76 to

correct for distortion. If the vein is displaced laterally away from the bronchial wall, the thickness of the latter is added to the measurement, since in most cases this thickness coincides with the medial portion of the vein.

In a case of obstruction of the superior vena cava by a giant metastatic mediastinal mass, the azygos vein measured 14 mm. (seated) and 18 mm. (recumbent). In thrombosis of the inferior vena cava due to migratory thrombophlebitis, the values were 11 mm. (seated) and 23 mm. (recumbent). In this patient, venography showed a collateral circulation via the epigastric veins to the mammary veins and via the deep pelvic veins to the azygos vein. A large anastomosis was also present between the left spermatic vein and the superior vena cava. Among 23 patients with portal hypertension, esophageal varices were found 11 times and the mean value, with or without varices, was 9.3 ± 1.8 mm. (seated), and 18.1 ± 2.4 mm. (recumbent). The arithmetic difference is not significant but most patients without varices showed values ranging from 9 to 11 mm. (seated); whereas, those with varices showed a larger number of normal values (seated) and also a larger number of increased values (seated). In other words, the presence of esophageal varices accounts for a greater distributional spread, partly depending on the extent of the portopulmonary (veno-arterial) shunt. The most difficult cases to evaluate were the cardiac patients with varying causes for right- and left-sided failure, different stages of compensation and variations in the types of shunts, such as the pulmonocaval (arteriovenous) shunt, which may be associated with left-sided insufficiency.

A considerable amount of knowledge concerning the distribution and extent of collateral veins in a variety of disturbances affecting the cardiac, portal and caval circulations can be gained by investigating the width of the azygos vein. A comprehensive and clear schematic drawing is included in this article to show the anatomic relationships of the venous systems and to indicate the development of the many types of collateral pathways. The following table lists the measurements obtained in the patients who comprised the major portion of this study.—*J. Zausner, M.D.*

	Azygos (seated)	Standard Deviation	Difference (seated and recumbent)	Cases
Normal	6.8 mm.	± 1.5	9.1 ± 2.2	18
Portal Hypertension	9.3 mm.	± 1.8	8.8	23
Right-Sided Insufficiency	8.3 mm.	± 1.5	4.2	15
Left-Sided Insufficiency	12.6 mm.	± 2.2	5.1	14
Compensated and Right-Left Insufficiency in Equilibrium	8.6 mm.	± 1.4	8.1	10

Lissner, J. Der Wert des Pneumomediastinums bei der Differentialdiagnose mediastinaler Erkrankungen. (The value of pneumomediastinum in the differential diagnosis of mediastinal diseases.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1959, *91*, 445–456. (From: Röntgenabteilung der Medizinischen Universitätskliniken, Frankfurt/Main, Germany.)

Pneumomediastinum may be of great help in determining the site of origin of mediastinal tumors as well as their size and attachments to neighboring structures. The presence of a stalk-like process extending craniad from an anterior mediastinal tumor is characteristic of thymomas. Pericardial cysts and abnormalities of the heart and great vessels can more easily be differentiated from other mediastinal masses by means of pneumomediastinum. Adhesions to mediastinal pleura or vascular structures can also be quite readily recognized and thus help in the preoperative evaluation. Similarly, central bronchogenic carcinoma and hilar metastases can be easily delineated.

One of the author's patients, who complained of dysphagia, had changes on barium swallow suggesting a leiomyoma. Pneumomediastinum disclosed a bronchial cyst with a short broad process originating from the left main bronchus. Tiny curvilinear calcifications within the mass also indicated the bronchial origin of the mass.

Some diagnostic difficulties in borderline cases may be resolved by kymographic studies to distinguish vascular from nonvascular tumors.—*J. Zausner, M.D.*

Abdomen

Frye, Thomas R. Villous adenoma of the sigmoid colon. *Radiology*, July, 1959, *73*, 71–75. (Address: Ohio State University Health Center, Columbus, Ohio.)

Benign epithelial tumors of the colon may be divided into two categories: the villous adenoma and the adenomatous polyp. Eighty per cent of these

tumors occur in the rectum and sigmoid. The adenomatous polyp occurs in about 7 per cent of all adults. The villous adenoma is rare, occurs singly, and constitutes from 0.1 to 6.6 per cent of all rectal and sigmoid lesions.

One case of villous adenoma of the sigmoid colon is reported by the author. The most prominent and characteristic symptom was the profuse discharge of large amounts of mucus, separate from the stool. Other complaints were anorexia, nausea, vomiting and weakness. There was no history of rectal bleeding, diarrhea or pain. Dehydration and electrolyte imbalance were present on admission. The barium filled colon showed a sharply demarcated mass into which extended linear streaks of barium apparently between the mucus covered folds and villi of the tumor. Following evacuation, the tumor appeared reduced in size, with a rough, serrated, feathery margin. This reduction in size is attributed to the expulsion on evacuation of the large quantity of mucus produced and collected within the tumor.

The lesion was removed surgically. The pathologic specimen was a large papillary mass that extended 12 cm. longitudinally and encircled the bowel without anatomic obstruction. The cut section showed mucosal proliferation and individual papillary fronds that measured 3 to 5 mm. in length and 0.5 to 0.1 mm. in diameter. The villous mucus membrane was thrown up in broad undulating folds which were freely moveable over the muscular wall. The submucosa appared normal. No malignant changes were noted on microscopic sections.

Early diagnosis of these tumors is important. Various authors have stated that malignant transformation occurs in 20 to 100 per cent of the cases. Sunderland and Binkley found malignant transformation in 68.7 per cent of their cases, 39.5 per cent being invasive carcinoma. A marked tendency to recurrence after local removal has been noted. In one series, approximately one-third of the cases studied showed an additional benign or malignant tumor of the bowel.—*W. M. McBride, M.D.*

Nuvoli, Umberto. Bijodati, trijodati, esajodati nella fisiologia e nella patologia dell'apparto epatobiliare e nuove vedute sulla patogenesi dei processi colecistitici e della colica epatica. (Biiodates, triiodates and hexaiodates in the physiologic pathology of the hepatobiliary tract and new views on the pathogenesis of cholecystic processes and hepatic colic.) *Radiol. med.*, Sept., 1959, *45*, 817–833. (From: Istituto di Radiologia del Policlinico—Scuola di Radiologia, Scuola Medica Ospedaliera di Roma, Rome, Italy.)

The author lists the indications of the various contrast media used for cholecystography. He demonstrates the superiority of the biiodates to test the function of the gallbladder and to confirm the presence or absence of a cholecystic disorder. The examination with biiodate compounds should precede others, which may be considered complementary. In fact, gallbladders not opacified by means of biiodates, which are suspected of being pathologic will normally be visualized by either triiodates or hexaiodates.

On the basis of clinical and anatomic criteria, the author affirms that the diagnosis of calculosis does not prove the existence of a cholecystic disease. He has observed cases in which, despite the presence of biliary calculi, the gallbladder was entirely normal macroscopically as well as microscopically. In such cases, one may obtain normal visualization with biiodates.

The use of hexaiodates has demonstrated that in chronic cholecystitis there exists a biliary stasis. It is evident that the basis of every pathologic manifestation is an inflammatory process, established by the elimination of pathogenic organisms through the bile

Utilizing hexaiodates it has also been noted that hepatic colic is the expression of a severe spasm of the entire biliary tract which is initiated at the level of the sphincter of Oddi and extends superiorly to involve the choledochus, cystic duct and the gallbladder.—*Anthony A. Blasi, M.D.*

Geffen, Abraham. An evaluation of orabilex (bunamiodyl), a new medium for cholecystography. *Radiology*, June, 1959, *72*, 839–843. (Address: Beth Israel Hospital, 10 Nathan D. Perlman Place, New York 3, N. Y.)

Orabilex, a new medium for cholecystography, has been given to 100 patients and the results have been compared to those reported for teridax and telepaque. This medium appears to be a safe and effective one, giving good visualization of the gallbladder.

Optimum dose evaluation showed a dose of 4.5 gm. to be fully as effective as a dose of 6 gm., regardless of the weight of the patient. All nonvisualized gallbladders were checked further with telepaque and showed the same findings. Eighty per cent of the patients showed no side effects at all and this appears to represent a significant improvement over other cholecystographic media. It is the author's opinion that orabilex in 4.5 gm. doses gives visualization comparable to telepaque in 3 gm. doses, with a notably lower incidence of side effects, and that it is superior to teridax on both scores.—*Donald N. Dysart, M.D.*

Tuqan, Nimr A., and Saleeby, George W. Primary reticulum-cell sarcoma of the spleen; report of a case presenting as a cyst. *Ra-*

diology, June, 1959, *72*, 868–871. (Address: N. A. Tuqan, American University of Beirut, Beirut, Lebanon.)

The authors present their observations of one case of primary reticulum-cell splenic lymphoma. The neoplasm was shown on the roentgenographic studies as a large impinging spherical mass located in the left side of the abdomen. On exploratory laparotomy, an enlarged spleen containing 3,500 cc. of hemorrhagic fluid was surgically removed. The gross pathology revealed a spleen which contained a large cyst of the superior pole and a solid tissue mass of the lower pole. On histologic examination, the neoplasm was diagnosed as a reticulum-cell sarcoma. It probably arose as a giant follicular lymphoma. There was no evidence of extrasplenic tumor at the time of surgery. Expiration of the patient eighteen months later was preceded by anemia, recurrence of tumor in the region of the left diaphragm, pleural effusion, and signs of rib destruction.—*W. M. McBride, M.D.*

Genitourinary System

Piemonte, M., and Magno, L. Mezzi di contrasto bijodati o trijodati? Ulteriori richerche sul meccanismo di eliminazione renale dei mezzi di contrasto per urografia. (Biiodate or triiodate contrast media? Further investigation on the mechanisms of renal excretion of urographic contrast media.) *Radiol. med.*, Aug., 1959, *45*, 744–785. (From: Istituto di Radiologia della Università di Milano, Milan, Italy.)

The authors review the chemistry of the contrast media and the modality of their renal excretion with special attention to biiodates and triiodates, particularly in the light of their dangers in urography on the basis of physiopathologic and statistical considerations. They note that there is no significant difference in this respect between the various contrast solutions commonly in use. According to acceptable statistical studies, the incidence of mortality is 6.6 for every million urographic examinations.

The authors' data include 64 cases in which a determination of the amount of iodine eliminated in the urine has been made. A comparison of the iodine eliminated and the volume of the urine excreted with the intensity of opacification of the urinary tract brings the authors to the following conclusions: (1) A direct ratio beteween the quantity of iodine eliminated and the concentration of urinary iodine with the intensity of opacification of the urinary tract does not exist; (2) there is a direct ratio between the volume of the urine eliminated during the examination and the intensity of contrast in the urinary tract; and (3) there is no essential difference in visualization between the biiodate and triiodate urographic contrast media.—*Anthony A. Blasi, M.D.*

Hope, John W., and Michie, Alexander J. Hydronephrosis following retrograde pyelography. *Radiology*, June, 1959, *72*, 844–849. (Address: J. W. Hope, 1740 Bainbridge St., Philadelphia 46, Pa.)

Prior to the present study, the authors, using the renal clearance technique, had found that ureteral catheterization disturbed kidney function. In an attempt to graphically demonstrate this physiologic disturbance, 14 children were selected for intravenous urography, to be followed by retrograde pyelography and finally by repeat intravenous urography within sixteen hours of the retrograde study. The second urogram showed abnormalities not previously present ranging from minimal stasis in the ureter to severe dilatation of the kidney with delayed appearance of the contrast medium. These changes were present in 9 of the 14 children studied, being severe in 1, moderate in 4, and minimal in 4.

The degree of renal impairment is probably secondary to trauma which causes edema of the ureteral mucosa, producing partial obstruction to urinary flow; consequently, one would expect greater impairment in children because of the smaller diameter of the urethra and ureters. It has been shown that the changes are transient, but urinary stasis of any degree predisposes to infection and should be avoided. An attempt should be made to perfect the technique of intravenous urography, since it is preferable to retrograde pyelography, and every effort should be made to arrive at a diagnosis without resorting to ureteral catheterization.

Three illustrative case reports are presented.—*W. H. Jarvis, Jr., M.D.*

Lemaitre, Guy, and Jules, Raymond. La déférento-vésiculographie: application au diagnostic de la tuberculose séminale. (Deferento-vesiculography: its use in the diagnosis of seminal tuberculosis.) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1959, *40*, 535–544. (From: Service de Radiologie [bloc Est] et Clinique Urologique de la Cité Hospitalière, Lille, France.)

This technique is not widely practiced because of the surgical approach needed to perform it. It has been done to study male sterility, nonspecific vesiculitis, changes in seminal vesicles secondary to prostatic lesions and seminal tuberculosis.

In the 18 cases studied by the authors, all the patients had proved renal or epididymal tuberculosis. In 8 cases, this examination was prompted by a low ureteral stenosis discovered on pyelography. In 6 other cases, the findings by rectal examination made this investigation mandatory.

In 12 of the 18 cases, deferento-vesiculography was definitely abnormal; 6 of these were considered negative after rectal examination. Following is an analysis of the authors' findings.

Vas Deferens. There was blockage in 3 cases in the preampullary portion with dilatation of the opacified vas deferens. Irregular caliber of the vas deferens was demonstrated in 2 cases, with multiple narrowings in the inguinal portion. Ulceration was seen in 1 case, appearing like a diverticulum perpendicular to the lumen of the vas deferens. One case presented multiple small diverticula along both vasa deferentia. This finding has never before been published and its significance awaits pathologic explanation.

Ampulla. There was bilateral dilatation of the ampulla with irregular margins in 2 cases, dilatation with normal contour in 4 cases, a large cavity of the proximal ampulla in 1 case, and narrowing and straightening of the ampulla in 2 cases. (In another, its tortuous course was preserved.)

Seminal Vesicle. Dilatation with normal contour was present in 2 cases and with jagged margins in 2 cases. Partial or nearly complete amputation was demonstrated in 2 cases, the remaining opacified portion showing irregular borders. There was filiform narrowing of the vesicle due to atrophy in 2 cases and lacunar shadows in the vesicle in 3 cases.

The authors consider the following signs as very suspicious of tuberculous involvement: blockage in the intrapelvic portion of the vas deferens with ulceration along the canal or at the point of blockage; dilatation of the ampulla, with grossly irregular contour, or ulcerations; and dilatation of the seminal vesicle with markedly irregular borders and loss of tubular structure or partial amputation of the vesicle with irregular contour of the remaining portion.—*H. P. Lévesque, M.D.*

Skeletal System

Fournier, André M., and Jullien, Georges. (Marseille, France.) Aspects radiologiques de la maladie des caissons. (Radiologic aspects of caisson disease.) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1959, *40*, 529–534.

This disease is a constant threat to divers and tunnel workers. The clinical picture is pleomorphic and often deceiving. With the exception of the "pressure blow" well known in occupational medicine, caisson disease offers no definite symptomatology. Its signs are varied; its evolution capricious; and its treatment uncertain. There are four important roentgen signs:

(1) *Areas of Demineralization.* (a) Demineralization patches are localized, like all the changes to be discussed later on, to the shoulder and the hip. They occur in the spongiosa of the humeral head (seldom in the glenoid process) and in the cervicotrochanteric region of the femur (rarely in the cotyloid region). They may appear as large, nonhomogeneous, ill-defined radiolucent zones. More often, small, rounded, sharply delineated lacunae are seen, sometimes coalescent, that may later form a large demineralized area. These signs of demineralization are not pathognomonic, unless substantiated by the history of the patient and his occupation. The typical localization in the hip or shoulder should suggest the possibility of caisson disease, especially in a young adult showing a well preserved mineral content elsewhere in the bones. (b) Geodes, round or oval, show a well-delineated opaque margin formed by reactive condensation of the surrounding bone. They are sometimes multiple and aggregated and vary in size from a few millimeters to 1.5 cm. They are not accompanied by degenerative arthrosis until the terminal phase. These geodes are not as characteristic as the demineralized patches since microgeodes are frequently seen in rheumatoid arthritis, gout, certain osteitises, and some reticuloses (Hodgkin's disease, sarcoidosis). However, in caisson disease, they are a little larger, less numerous, and their opaque margins are less constant. Again, the location of these geodes in the upper part of the femur and humerus and the appearance of the surrounding bony structure should be weighed in the differential diagnosis.

(2) *Bone Condensation due to Osteosclerosis.* (a) The compact type is the most characteristic with dense, rather homogeneous, compact bone replacing the spongious subcortical bone, and producing a dense zone along the articular border, the distal side being sharp and irregular. This hemispheric condensation may later on progress towards the center of the humeral head and produce a depressed triangular shadow. (b) The trabecular type, produced by thickened trabeculae, is usually seen in the metaphyseal area of the femur close to the lesser trochanter. The lesions are disposed in a criss-cross manner with small, scattered, more or less coalescent islands of osteosclerosis.

(3) *Sequestration.* A crescentic or large triangular portion of the subchondral bone becomes sequestrated and later on is pushed inside the head of the femur or humerus.

(4) *Coxarthrosis with Osteophytic Formations.* This condition shows extensive destruction and important remodeling.—*H. P. Lévesque, M.D.*

Burman, Michael. Osteotomy of the spine; formation of a wedge vertebra in the hyperextension gap. *Radiology*, July, 1959, *73*, 104–106. (Address: 130 E. 67th St., New York 21, N. Y.)

The author states that Marie-Strümpell disease or ankylosing spondylitis produces two major skeletal defects: fixation of the costovertebral joints and flexion deformities of the spine. The hip joints may be involved, adding to the flexion deformity of the spine. The affected person may see nothing but the ground when he walks. The etiology is unknown; there is no specific treatment. Roentgen therapy to the diseased spine relieves pain but may cause leukemia.

Attempts at straightening the spine by closed manipulation invite paraplegia or, possibly, rupture of the aorta. Surgical correction of the fixed costovertebral joints has not been attempted. However, two types of surgical procedures have been used: (1) the indirect or bypassing extension osteotomy of the upper end of each femur (Milch), which seems to straighten the back, and (2) direct osteotomy of the spine, especially in the "parenthesis" form described by Sotelo-Ortiz. A surgical procedure used by Smith-Petersen and his colleagues is a definitive open osteotomy of the spine. This is performed at the site of greatest deformity in the spine, usually at the midlumbar level. A large wedge of bone with the posterior base is removed, including the adjacent spinous processes, the laminae, and the fused apophyseal joints. This procedure may be done at one or two levels, seldom more than two. When the surgery is done at one level, as in the technique of Adams, the hinge joint is the posterior part of the intervertebral disk. The upper vertebra tilts on the lower one. The hyperextension gap fills with blood and fragments of fibrous tissue. The anterior longitudinal ligament breaks as the spine is hyperextended. It is not necessary to fuse the spine. There is no gap when the correction is small or done at more than one level, as in McMaster's cases. However, Lichtblau and Wilson found that correction was achieved at only one interspace, and no additional correction was gained by liberating two or three interspaces.

Ossification may occur in the hyperextensive gap. This creates a new wedge vertebra. The author presents 1 such case, and refers to 1 case reported by La Chapelle and 1 case by Sotelo-Ortiz. The tension of the hinge joint discourages new bone formation. However, ossification and formation of a wedge vertebra may be due to tension of the hinge joint being offset by compression of a straight spine, together with the fact that new bone formation is inherent in the disease.—*W. M. McBride, M.D.*

Papavasiliou, C. G., and Speas, C. J. Fracture of the hyoid bone. *Radiology*, June, 1959, *72*, 872–874. (Address: C. J. Speas, Oak Ridge Hospital, Oak Ridge, Tenn.)

Fractures of the hyoid bone are the result either of direct trauma, as strangulation, car accidents or gunshot wounds, or of muscular action, as in sudden hyperextension of the neck or forcible deglutition. Early bony union between the body and the greater cornu or anomalous ossification is a predisposing factor. The condition may be serious, with severe pain, dysphagia, dysphonia, or severe dyspnea. Fatal asphyxia frequently follows this injury unless relieved by tracheotomy. Roentgen-ray demonstration of a fracture of the hyoid bone requires no special views. The true lateral and oblique views usually demonstrate the bone satisfactorily. The most frequent site of fracture is the junction of the body with one of the greater cornua.

Two cases of fracture of the hyoid bone diagnosed clinically and roentgenologically are reported by the authors. Both were due to direct trauma, one by a single buckshot and the other by a direct blow. Dyspnea was the most prominent symptom necessitating the insertion of a tracheotomy tube.—*Arno W. Sommer, M.D.*

Fischgold, H., Bernard, J., and Baudey, J. (Paris, France.) Les cartilages épiphysaires de l'enfant. (Epiphyseal cartilages of children.) *J. de radiol., d'électrol. et de méd. nucléaire*, Aug.–Sept., 1959, *40*, 429–443.

Are epiphyseal cartilages of children visible on roentgenograms? Various authors affirm, deny, or are ambiguous on the point; but radiologists, being accustomed to reporting brilliant contrasts, do not describe them even when they are visible. Yet cartilage is denser than muscle and can often be visualized with a non-screen technique, particularly with LogEtron. The usual factors are 45–60 kv. peak, 200–300 ma., and 0.3 second or less. As fat constitutes a contrast material *in situ*, best visibility occurs where fat and cartilage are juxtaposed, as about the patella, popliteal space and os calcis.—*Frank A. Riebel, M.D.*

Forster, E., Molé, L., and Peter, R. Lésions du genou (confrontations radiocliniques). (Lesions of the knee [radiologic and clinical comparisons].) *J. de radiol., d'électrol. et de méd. nucléaire*, Aug.–Sept., 1959, *40*, 418–428. (From: Service de Chirurgie et Service de Radiologie, Centre hospitalier L.-Pasteur, Colmar, France.)

In trauma of the knee, present therapeutic conceptions call for rapid surgical treatment of a meniscus or ligamentous tear. This necessitates minute clinical study and a well directed roentgenologic investigation.

The authors inject 5 cc. of 50 per cent diodine into the knee joint and follow it with 30 to 40 cc. of air. Under fluoroscopic control and using a grid they take serial spot roentgenograms, 8 views for each meniscus in varying degrees of rotation and in forced positions. The latter are insufficiently used generally and represent an important part of this procedure, which nearly always ensures definitive diagnoses of ligamentous tears. Arthrography is particularly valuable for tears in the posterior horns of the menisci, which remain invisible even in arthrotomy, and for post-traumatic laxities.—*Frank A. Riebel, M.D.*

Blood and Lymph System

Luke, H. A. Experiences with Seldinger's technique of arterial catheterization. *Proc. Coll.*

Radiologists, Australasia, June, 1959, *3*, 1–16. (From: Department of Diagnostic Radiology, The Alfred Hospital, Melbourne, Australia.)

This paper was prompted by the ever increasing need for accurate, safe arteriography as an adjunct to surgery. It is concerned with the results of the use of Seldinger's technique of arteriography in the hands of the author.

Previous methods of arteriography (dos Santos, Robb, Jonsson, Nuvoli, Radner, Euler, Burford, Freeman) all had various disadvantages, involving either lack of control over the point of puncture (direct injection) or the sacrifice of a vessel and scar formation (direct exposure). Seldinger's technique is apparently an outgrowth of a technique suggested by Pierce and involves the use of a polyethylene catheter and guide wire.

The method consists of inserting a needle into the femoral or brachial artery. Over this needle there is a closely fitting cannula with thin walls. After the cannula has been passed into the lumen of the vessel, the needle is removed and is replaced by a long, flexible guide wire made from stainless steel. The cannula is removed and over the guide wire is passed a polyethylene catheter, the tip of which is shaped to fit the guide wire tightly. The remainder of the catheter can be larger in diameter than the tip, as the tip serves as a dilator. Once the catheter is well in the lumen, the guide wire is removed and the catheter is pushed along to the proper level. This allows for pinpoint injection of the contrast medium at the desired level and ensures the impossibility of injecting intramurally, thus conserving the vessel. By means of sharp or mild curves in the catheter, the tip can be easily guided into various vascular orifices at will.

In this series, 60 per cent urografin or 50 per cent hypaque was used as the contrast medium. If the leg, pelvic or abdominal vessels were to be examined, the catheter was usually inserted through the femoral artery. If the carotid arteries were to be studied, the catheter was placed directly in the carotid artery. The ascending aorta, arch and upper descending aorta could be examined from either site.

Each examination poses its own problems but can be dealt with by simple changes in the length and shape of the catheter and by adjustment of the dosage of the contrast medium. It was found that the use of image intensifiers and opaque catheters greatly reduced problems of the procedure, *i.e.*, sterility, pinpoint injection of medium, etc.

Twelve cases are cited, with accompanying roentgenographic reproductions which admirably illustrate the various uses to which this technique may be put. No instance of complication was found.—*William C. MacCarty, Jr., M.D.*

Vaughan, B. F. Experiences in aortography by femoral artery catheterisation with the Seldinger catheter. *Proc. Coll. Radiologists, Australasia*, June, 1959, *3*, 17–23. (From: Department of Radiology, Royal Perth Hospital, Perth, Western Australia.)

The author reports on experiences with aortography using femoral artery catheterization with a Seldinger catheter, emphasizing some of the difficulties encountered and citing several successful applications where aortography by this method proved to be of great value.

A 205 gauge catheter was used in all patients, with right femoral catheterization preferred. Four roentgenograms were routinely taken—near the end of the injection and at one second intervals thereafter.

Two difficulties encountered which made it impossible to advance the catheter were thrombus at the aortic bifurcation and abnormal tortuosity of the iliac arteries. The danger of attempting to insert a long length of guide wire in the presence of the latter condition is noted, and a case is cited in which extravasation of the contrast medium resulted from external iliac artery penetration. In a second case, contrast medium was injected into a thrombus present inside a large abdominal aneurysm with subsequent rupture of the aneurysm two days later.

Several successfully catheterized cases are presented, with 3 illustrating the value of aortography in planning vascular surgery. The possibility of a tortuous abdominal aorta being clinically diagnosed as an aneurysm is mentioned.—*William C. MacCarty, Jr., M.D.*

Canter, Jerome W., Kreel, Isadore, Rosenthal, William S., and Baronofsky, Ivan D. The effect of local factors on the portal venous pattern. *Surg., Gynec. & Obst.*, Dec., 1959, *109*, 729–734. (From: Division of Experimental Surgery, Department of Surgery, and Division of Gastroenterology, Department of Medicine, The Mount Sinai Hospital, New York, N. Y.)

Portal venography has been a valuable aid in the investigation of portal hypertension and the portal venous system. The transplenic route of injection of the contrast medium has been of particular value in clinical work. The enlarged, fibrotic spleen of a patient with cirrhosis of the liver lends itself well to percutaneous splenoportography. In the dog, with a small, friable, mobile spleen, it was found expedient to transplant a portion of the spleen to a subcutaneous pocket in the anterior abdominal wall.

In a series of experiments on dogs, an attempt was made to evaluate the role of certain local factors, other than liver disease *per se*, in the roentgenologic appearance of the portal venous system. Sixteen dogs were used and each had the tail of the spleen transplanted to the subcutaneous layer of the abdominal wall. Splenoportography was then carried

out to establish a normal pattern. The thoracic inferior vena cava, above the diaphragm, was partially constricted in 13 animals to induce the formation of ascites. Artificial ascites was created in 3 dogs by the infusion of saline into the peritoneal cavity. All dogs had splenoportograms for comparison with previous normals. Certain changes in the venous pattern were always present and it became possible to differentiate between obstruction to venous outflow with hepatic congestion and that change due merely to the ascites and increased intra-abdominal pressure.

The article is well illustrated with roentgenographic reproductions showing the variations in the portal venous pattern.—*Richard E. Kinzer, M.D.*

Malamos, B., Moulopoulos, S. D., and Sarkas, A. Lymphadenography: its uses in haematology. *Brit. M. J.*, Dec. 19, 1959, *2*, 1360–1361. (From: Department of Clinical Therapeutics, University of Athens, Athens, Greece.)

The purpose of the present investigation was to visualize by roentgenography not only the lymph vessels but particularly the lymph nodes in several hematologic diseases. The main use of lymphadenography is to help distinguish between benign and malignant lymph node diseases. In normal cases the lymph nodes are small, with regular contour and equal density. In malignant diseases they may be larger and they are irregularly outlined, often with one or multiple defects at the border of the shadow, with a "worm-eaten" appearance and an unequal density in several parts of the lymph node.

Characteristic changes were observed in all cases of malignant hematologic diseases such as leukemia, lymphogranuloma, and lymphosarcoma; however, lymphadenography cannot as yet differentiate with certainty between these malignant conditions. It is suggested that the method could be of great help in cases lacking apparent involvement of the peripheral lymph nodes.

Up to now this method has not given any false-positive or false-negative results. Attention should be paid, however, to aged patients, in whom the lymph node contour can be irregular in normal cases. It is of interest that a positive result can be obtained even if the lymph nodes are not considered abnormal on palpation.

The technique of injection of the lymph nodes is described, and reproductions of roentgenograms of injected lymph nodes are included.—*D. S. Kellogg, M.D.*

General

Copping, D. E. Cockayne's syndrome (syn. heredo-familial multi-dystrophic dwarfism). *Proc. Coll. Radiologists, Australasia*, June, 1959, *3*, 52–54. (From: Princess Margaret Hospital, Perth, Western Australia.)

Cockayne's syndrome is characterized by dwarfism, hearing defects, lack of subcutaneous fat, hypersensitivity of the skin to sunlight, arrested mental development and pigmented retinal degeneration. The author describes the roentgenologic findings in 3 siblings of an Australian family with this syndrome.

Cases reported to date have all demonstrated generalized thickening of the calvarium, most evident in the frontal and occipitoparietal regions. In addition to this, the present cases had short, broad metacarpals and phalanges, similar to those in achondroplasia but with normal carpals and forearms. Each also had ivory-dense epiphyses of some of the terminal phalanges as well as epiphyseal notching of the anterior margins of the upper and lower surfaces of the vertebral bodies. It is uncertain as to whether these roentgenographic findings, in addition to a thickened calvarium, are a part of the syndrome or individual variations in this family.—*William C. MacCarty, Jr., M.D.*

Etter, Lewis E.; Krammer, Adolph G.; Dattoli, John A.; Cember, Herman; Griffith, Charles R.; and Cross, Lawrence C. Radiation reduction by combined copper and aluminum filtration in radiography. *Radiology*, June, 1959, *72*, 863–867. (Address: L. E. Etter, 3601 Fifth Ave., Pittsburgh 13, Pa.)

There has been much concern among both medical men and laymen as to the amounts of radiation exposure obtained by patients coincident with routine diagnostic roentgenographic procedures. Various methods of reducing patient exposure are available. These include long focus film distance, small cones, diaphragmed beams, fast screen and film, and accelerating chemicals for processing of film.

The present article describes experimental studies, some of which were done on a mannikin simulating a human body, directed towards reducing radiation exposure by filtering roentgen-ray beams with various combinations of aluminum and copper filters. Using a combination of 0.375 mm. of copper and 3.0 mm. of aluminum (90 kv., 4 ma.) for example, the authors have demonstrated an 88 plus per cent reduction in dosage at the table top and to the patient's skin during routine conventional fluoroscopic examinations. Similarly, in addition to measurements on the mannikin, they made direct measurements adjacent to the gonads in male patients during cinefluorography. In this procedure, at 100 and 120 kv. (1 ma.), they found that scanning of the patient, as for a routine gastrointestinal study, with no beam filtration, resulted in very high gonadal doses. When 0.375 mm. of copper and 1.5 mm. of aluminum were added, the average gonadal dose was only 5 mr per minute for the same procedure. Using only 3 mm. Al filtration, the dose averaged 8 mr per minute.

Of greatest importance is a consideration of depth doses in the various tissues and bone marrow. With the above combination of copper and aluminum, in spite of the higher kv. and ma. factors, there is a lowered net depth dose. This is because, with a higher quality beam, even though the percentage depth dose is increased, the skin dose is so low that the net dose to the deep tissues is actually reduced.

Comparative graphs and charts are presented to show the reduction of doses to the skin, gonads and bone marrow in routine roentgenography of the skull, abdomen and chest using various thicknesses of aluminum-copper filters and various combinations of commercial type screens and films. In combination with the previously mentioned methods, the use of filtered beams, as described, is recommended by the authors as an aid in reducing patient exposure during routine diagnostic roentgenologic procedures.—*Z. Petrany, M.D.*

Kazan, B., and Godfrey, E. W. Experimental results with the panel x-ray amplifier. *Radiology*, June, 1959, *72*, 855–862. (Address: B. Kazan, Hughes Research Laboratories, Culver City, Calif.)

An experimental panel roentgen-ray amplifier making use of electroluminescent and photoconductive materials is described. This solid-state amplifying panel approximately ½ inch thick and 12 inches square is positioned and viewed in a manner similar to conventional fluoroscopic screens. Roentgen rays falling on the photoconductive layer increase its conductivity, thus modulating the flow of A.C. current through the corresponding layer of electroluminescent material, which then emits a bright pattern of light. The image thus produced is approximately 100 times brighter than conventional fluoroscopic screens excited at the same roentgen-ray level.

With the roentgen rays stopped, the output decays slowly over a period up to thirty seconds providing temporary storage of the picture. Because of this long decay period, moving images cannot be viewed. This prolonged image would be an advantage, however, in setting up portals for therapy purposes. Other possible uses and improvements which might be made to extend the usefulness of such panels are discussed.—*John S. Alexander, M.D.*

Marin Gorriz, E. F. J. Notas preliminares sobre la roentgenografia con 200 kilovoltios. (Preliminary observations on 200 kv. roentgenography.) *Acta iber. radiol. cancerol.*, Jan.–Mar., 1959, *14*, 1–17. (From: Cátedra de Terapéutica Física, Facultad de Medicina de Zaragoza, Zaragoza, Spain.)

For some time the author has been using a 200 kv. apparatus for roentgenography, with excellent results. He uses a therapy roentgen-ray tube with a target of 2 mm., and the radiation is filtered through 2 mm. Cu, giving a half value layer of 2 mm. Cu. The milliamperage can be varied from 2 to 20 but he finds it more convenient to use from 2 to 8 ma. in order to have greater control over the exposure time, which usually amounts to several seconds, but never more than 10.

The main usefulness of high voltage roentgenography is the diagnosis of diseases of the chest, pharynx, larynx, and facial air sinuses. Comparing his material with that of other investigators who have published their experience with supervoltage roentgenography in the 1,000 to 2,000 kv. range, the author feels that both techniques yield about the same results and that roentgenography with 200 kv. has the advantage of requiring only equipment easily available and usually present in all radiation therapy departments.—*F. Comas, M.D.*

Policard, A., Collet, A., and Pregermain, S. Innocuité des poussières de houille pour le protoplasma cellulaire; démonstration par microscopie électronique. (Innocuousness of coal dust to cellular protoplasm; demonstration by electron microscopy.) *Presse méd.*, Dec. 25, 1959, *67*, 2263–2265. (From: Centre d'Etudes et Recherches des Charbonnages de France, Groupe Poussières-Silicose, Paris, France.)

The toxic effects of silica and coal dust were studied by introducing a mixture of them intratracheally into the lungs of rats. Seven days later, ultra-thin sections (0.04 microns) were studied with an electron microscope. In this short time, modification of cytoplasmic infrastructure was seen about the silica particles, apparently from denaturization of the constituent proteins, while no such changes appeared about the coal particles.

The authors conclude that in anthracosis disorders depend on obstructive factors only, whereas toxic effects develop from the beginning in silicosis.—*Frank A. Riebel, M.D.*

Freyer, B. Über Wachstumsstörungen des Skelettsystems durch ein strahleninduziertes infantiles Myxödem. (Growth disturbance of the skeletal system in radiation induced infantile myxedema.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1959, *91*, 305–311. (Address: Waldmeisterstrasse 2, Berlin-Dahlem, Germany.)

The author presents a case of a twenty-nine year old man who developed myxedema twenty-three years ago, after having received roentgen therapy for papillomatosis of the vocal cords. This treatment, over a period of one year, was given only after numerous operations failed to arrest the condition. It was estimated that with the physical factors used (120

kv., half value layer 0.22 mm. Cu, 40 cm. focal skin distance, two lateral and one anterior 6×8 cm. portals) a depth dose of 2,010 r was delivered to the thyroid gland and 1,540 r to the vertebral column. The irradiated skin showed atrophy and telangiectasia. In spite of the above dose, recurrent disease necessitated further surgical intervention.

The patient was obese, had a dry skin and a childish voice. There was no uptake of I^{131}. The total cholesterol was increased to 463 mg. per cent, with the ester to 264 mg. per cent. Serum calcium was 8.63 mg. per cent and inorganic phosphorus was 5.85 per cent.

Three types of injury were noted: (1) arrest in growth of those vertebrae in the direct path of the irradiation; (2) radiation induced decrease of function of the thyroid and parathyroid glands, which (3) depressed the growth of the entire skeleton as the result of the production of a secondary infantile myxedema.—*Samuel Richman, M.D.*

RADIATION THERAPY

PISANI, G., and MALASPINA, A. Eighteen cases of cancer of the oesophagus treated with telecobalt-therapy. *Panminerva med.*, Sept., 1959, *1*, 201–205. (Address: G. Pisani, Via Giulietti 11, Novara, Italy.)

Eighteen cases of carcinoma of the esophagus were treated from July, 1957 to September, 1958 with a Picker-Barazzetti Co^{60} unit of 1,050 curies. Fifteen of the cases were males. The ages were from forty-eight to seventy-five years. All of the patients had radiologic evidence of carcinoma of the esophagus and 4 cases had confirmatory biopsy evidence. The lesion was located in the upper third of the esophagus in 3 cases, in the middle third in 9 cases, in the lower third in 4 cases and in 2 cases it was at the cardio-esophageal junction.

The patients were treated either by the static or kinetic methods. Static opposing fields were usually used for palliation. Otherwise, 3 fields were used with 1 anterior and 2 posterior fields with converging beams. Rotational therapy was used for mid- or low lesions of the esophagus and 2 opposing 120° pendulations were used for lesions in the upper esophagus.

The location of the lesion was plotted accurately from roentgenograms and the surrounding structures were diagrammed from anatomic charts. Considerable care was taken in planning. Consideration had to be given to the degree of amplitude of pendulation and in rotation therapy to the center of rotation in those cases treated with kinetic therapy. Isodose curves were made and the fields were drawn on the patient.

A dose of 6,000 to 6,500 r was given in 4 to 5 weeks, with a daily dose of 150 to 200 r. Frequently, 4,000 to 4,500 r induced retrosternal pain and burning on deglutition and, if the symptoms were too severe, the treatment was divided into 2 cycles with a 45 to 60 day interval and a total dose of 7,000 r. Two patients received in 2 cycles 8,600 r and 9,000 r respectively. Palliation treatment was given to improve the caliber of the lumen and a daily dose of 100 to 150 r was given for 40 to 50 days, reaching a total dose of 4,000 to 5,000 r.

Eight of the 18 patients were living at the time of tabulation. Three had clinically recovered 9, 11 and 12 months respectively after treatment. Two of the 5 remaining cases had supraclavicular and low cervical lymph node involvement which disappeared after administrtaion of 5,500 r. The surviving patients showed a marked improvement before the end of treatment. The roentgenologic signs of the carcinoma disappeared in 7 cases. There were no general untoward effects of the treatments and there were no cases of radiation pneumonitis.

The authors believe that telecobalt therapy is indicated in the treatment of carcinoma of the esophagus since it produces uniform irradiation of the tumor with less injury to the surrounding tissues.—*Charles W. Cooley, M.D.*

MAGALOTTI, MARION F. Plaster casts for radiation therapy of oral carcinoma. *Radiology*, July, 1959, *73*, 100–103. (Address: Department of Radiation Therapy, Cook County Hospital, Chicago, Ill.)

The use of plaster casts for consistent beam localization in radiation therapy of oral carcinoma is described. This obviates the need for continued marking of the skin of the face or neck during the course of treatment. The desired port is outlined on the patient's skin with carbol fuchsin dye. It is not necessary for the outlined port to conform in size to the available cones. Resin plaster of Paris, in the form of rolls 4 inches wide, is molded to the contour of the patient and removed after five minutes. The carbol fuchsin dye skin markings are transferred automatically to the inner surface of the cast and these in turn are easily transferred to the outer surface through the use of straight pins thrust through the cast.

With the cast reapplied, therapy may be started with assurance of obtaining the same port at each treatment. The plaster cast presents no absorption problem as studies have consistently shown the transmission of radiation through the cast to be approximately 95 per cent.—*John S. Alexander, M.D.*

WILSON, WESLEY W. (Tampa, Florida). X-ray treatment of hemangiomas: a review of 500 cases and 15 years experience. *South. M. J.*, Nov., 1959, *52*, 1355–1358.

Five hundred infants with hemangiomas were treated with roentgen therapy from 1941 to 1957. The following factors were used: 50, 62 or 85 kv. depending on the thickness of the hemangioma, tar-

get distance of 15 cm. except in the very thick lesions when it was 24 cm., and inherent filtration of 0.5 mm. Al with no added filter. In the 200 patients treated between 1941 and 1952, 175 r was given per treatment at intervals of one week in the age group under five weeks for a total dose of 525 to 700 r. A dose of 200 r was given per treatment in those patients over five weeks of age at intervals of three weeks for a total of 800 to 1,000 r. The 300 patients treated from 1950 to 1957 received 200 r per treatment in the age group under five weeks at intervals of two weeks for a total of 400 to 600 r and in those over five weeks 240 r was given per treatment at intervals of three weeks for a total of 720 to 960 r.

In the first 200 patients treated, involution was complete by the end of the second year, in those infants who were treated before the age of five weeks. In those treated after five weeks of age, complete healing did not occur before two and one-half to three and one-half years of age. The 300 patients treated from 1952 to 1957 had an average involution time of 2 years and this was accomplished with a lesser total dose and in a shorter time.

Many of the patients have been followed for a fifteen year period and no evidence of tissue damage, bone destruction or other adverse effects has been manifest.

The author states that most of these vascular growths require treatment and the therapy indicated varies with the type of lesion, its location and the age of the patient. The object of treatment is to cause involution of the hemangiomas with the least possible injury to the skin. The chief danger is overzealous therapy. Port wine marks were not treated.

The author believes that roentgen therapy is particularly suited to the treatment of hemangiomas, especially the larger lesions, and that excellent therapeutic and cosmetic results are obtained.—*Charles W. Cooley, M.D.*

Du Sault, Lucille A. A simplified method of treatment planning. *Radiology*, July, 1959, *73*, 85–94. (Address: The Henry Ford Hospital, Detroit 2, Mich.)

The use of isodose curves which are independent of patient size and contour and which may be made up in advance for various combinations of fields is described. The proposed method, which is better adapted for use with supervoltage or cobalt therapy, appears to be extremely useful in rotational therapy. All doses are expressed as 100 per cent of the center or tumor dose, thus permitting rapid evaluation of a suggested treatment plan by using the previously prepared isodose curves. It is usually unnecessary to calculate the skin dose as dosage figures are based on the tumor dose.

The new type isodose curves extend indefinitely both above and below the center or 100 per cent point, which lies at a depth in tissue. As they are independent of the surface, isodose lines resulting from various combinations of fields, which will apply to any patient, may be made up in advance. In preparing the treatment plan for a patient, the body contour is taken and transferred to tracing paper. The region to be treated is outlined, and normal structures whose tolerance must be considered are indicated. This tracing is then placed over various isodose plans until the best location for the center and the best pathways for the beams are found.

This plan requires delivery of equal doses to the center. Since the center is always at the same distance from the source, the treatment time will depend on the thickness of tissue between the source and the center, which may be different for each position. The time will also depend on field size.

Examples of methods of calculating treatment times are given as well as the use of the tumor-air ratios in such calculations.—*John S. Alexander, M.D.*

Uhlmann, Erich M. Clinical experience with high-speed electrons in cancer therapy. *Radiology*, July, 1959, *73*, 76–84. (Address: Michael Reese Hospital, Chicago 16, Ill).

The author discusses the clinical application of therapy with electrons in the energy range between 20 and 40 mev. The information presented is the result of experience with a 45 mev. linear accelerator installed at the Tumor Clinic of Michael Reese Hospital.

The principal difference between high speed electrons and roentgen rays in the multimillion volt range lies in their depth penetration. In the case of electrons, the depth is sharply defined and mainly dependent upon the energy. The penetration of electrons in tissue, expressed in centimeters, can be obtained by dividing the mev. energy by 2. However, the depth at which a therapeutically useful dosage is delivered is best estimated by dividing the energy by 3. A percentage depth dose table for the 10 to 40 mev. range is presented along with isodose curves for 15, 25, and 35 mev. using a 10×10 cm. field. These curves were obtained from films sandwiched in unit density pressdwood.

Single field application of these isodose curves is demonstrated in carcinoma of the tongue and an instance of a solitary pulmonary metastasis from osteogenic sarcoma. An additional attractive feature of therapy with high speed electrons is the possibility of using angular or opposing fields, which reduces the skin dose to about 60 per cent of the depth dose. A multiple field technique is described in specific cases of carcinoma of the urinary bladder, the esophagus, and the nasopharynx.

An important factor in the therapeutic utilization of high speed electrons is the relatively small volume of tissue that is subjected to irradiation, even if the tumor is deep-seated. This factor alone may account

for the absence of systemic reactions.—*Edward B. Best, M.D.*

OELSSNER, WILHELM, PFEIFFER, JOACHIM, and BUTTENBERG, HELMUT. Osteoradionekrosen im Hüftgelenksgebiet. (Radiation osteonecrosis in the region of the hip joint.) *Strahlentherapie*, June–July, 1959, *109*, 200–210. (From: Universitäts-Röntgeninstitut, Leipzig, Germany.)

Of 909 patients treated for gynecologic tumors from 1952 to 1958, 25 developed radiation osteonecrosis about the hip. This incidence of 2.75 per cent is higher than usually reported, probably because of an organized follow-up study of all patients. This follow-up included periodic roentgenograms of the chest and pelvis, so that asymptomatic osseous changes were detected.

The osteonecrosis appeared as: zones of altered bony architecture in the femoral neck, ischium or pubis (Umbauzone), femoral neck fracture, femoral head necrosis, or acetabular necrosis with or without central protrusion.

Radiation therapy consisted of 3,000–3,800 r to the skin over each of three lower abdominal fields and three opposite dorsal fields (10×15 cm. ports with 180 kv., 0.5 mm. Cu filtration, 0.95 mm. Cu half value layer, 40 cm. focal skin distance, and 200–250 r administered per treatment). In addition, a perineal 10×15 cm. port received 1,200–1,600 r, and sometimes two lower gluteal 8×10 cm. ports and an intravaginal port were used.

No correlation between incidence of radiation osteonecrosis and the patient's constitution, weight or age was demonstrated. In every case, however, the femoral neck was in the direct radiation beam.

Treatment of the "fatigue fracture" alteration of osseous architecture should consist of immobilization and bed rest supplemented with administration of vitamin D and calcium. If symptoms do not subside, prophylactic nailing of the femoral neck is indicated. Some complete fractures can be treated similarly but, if any tendency to pseudoarthrosis develops, internal fixation by nailing is used. In the pseudoarthroses of long duration, nailing is often ineffective and arthrodesis must be resorted to.—*Henry G. Moehring, M.D.*

WINDEYER, B. W. (London, England.) Chordoma: President's address. *Proc. Roy. Soc. Med.*, Dec., 1959, *52*, 1088–1100.

Chordoma is a relatively rare tumor arising from notochord remnants. Vestigial remnants of notochord are occasionally seen in the vertebral bodies, and are represented in the intervertebral disks by the nucleus pulposus. Chordomas may develop anywhere along the notochordal axis; however, 90 per cent of these arise in the two ends of the vertebral column, in the spheno-occipital and sacrococcygeal regions.

Twenty-nine cases of chordoma are reviewed in this reported series. The youngest patient was eight and the oldest sixty-six years of age. Most patients were in the older age group, especially those with sacrococcygeal chordomas. Sex incidence was equal. The sacrococcygeal chordomas formed the largest group.

The typical chordoma is a slow growing, locally destructive, compressive and erosive tumor rather than an infiltrative one. It is of a solid gelatinous consistency. The great variation in histologic appearance is suggestive of the notochord in its various stages of development. There may be, for example, difficulty in differentiating chordoma from chondrosarcoma.

Local extension, proceeding at a slow pace, may allow the tumor to attain considerable size, causing interference in function or nerve involvement. Metastases, though rare, can develop via the blood stream or lymphatics. Clinical features of vertebral chordoma depend upon the site of origin and direction of spread and may be manifested by pain or nerve involvement. In sacrococcygeal chordomas, the earliest symptom was an aching pain followed by the appearance of a lump causing pressure or interference with micturition or bowel action.

On roentgenography, the chordoma is revealed by the irregular destruction of the affected bone as it burst through the cortex to form a projecting soft tissue mass. On expansion, it may carry small flecks of bone ahead of it and may develop calcification within it. Only 1 case in this series of 29 showed extensive calcification.

Complete surgical ablation is hardly ever possible due to the location of these tumors, as, for example, in the base of the skull and sacrum. Partial excision is an excellent palliative procedure and patients are kept going by repeated partial excisions.

Publications on chordoma often begin with a statement as to the radioresistance of chordoma, although some reports on the value of palliative radiotherapy are favorable. The author was impressed with the remarkable regression obtained in 2 cases treated with high doses of 200 kv. roentgen rays and has an optimistic and even enthusiastic view towards treatment of chordomas with supervoltage therapy. As a palliative measure, he feels that radiotherapy should be given in preference to partial operation in sacrococcygeal tumors. Supervoltage therapy provides the opportunity to deliver the necessary high dosage without excessive damage to the overlying and surrounding tissues.—*Bernard Loitman, M.D.*

BROWN, CHARLES H. Therapeutic principles in management of peptic ulcer; radiation therapy with cobalt-60. *Am. J. Digest. Dis.*, Dec., 1959, *4*, 1066–1072. (From: Department of Gastroenterology, The Cleveland Clinic

Foundation and The Frank E. Bunts Educational Institute, Cleveland, Ohio.)

Radiation therapy of the stomach has been used for many years in treatment of peptic ulcer, the purpose being to suppress gastric acid secretion. Various techniques and modalities of irradiation have been employed satisfactorily to achieve this effect including radium needles placed in an Ewald tube, intragastric balloons containing I^{131} or P^{32}, deep (250 kv.) roentgen therapy and, in recent years, supervoltage (Co^{60}) therapy.

This is a study of gastric irradiation employing Co^{60} teletherapy with a five year follow-up. Approximately 2,000 r depth dose was given through opposed anterior and posterior portals, with the upper two-thirds of the stomach outlined fluoroscopically. The use of cobalt allows delivery of the same radiation dosage in a shorter interval with fewer unpleasant side effects than is possible with 250 kv. roentgen rays. The therapeutic results are believed to be similar.

Of 72 patients, 48 had duodenal ulcer, 11 gastric ulcer and 13 marginal ulcer. All had medically intractable ulcers and were considered surgical candidates. In some, surgery was contraindicated because of cardiac conditions and other factors which made them poor risks.

Gastric analysis was the means of objective measurement of irradiation effect. Basal or fasting gastric secretory studies, as well as Histalog-stimulated gastric analyses were done. All patients showed a decreased gastric acid secretion and, in some cases, a gradual return of hyperacidity in from six to twelve months. Approximately 50 per cent still had a fasting achlorhydria twelve to twenty-four months following treatment. Initial symptomatic response and roentgen evidence of healing was good in all patients. Hyperacidity, symptoms and ulcers recurred in 3 patients.—*Bernard Loitman, M.D.*

Mathieu, Roger. On the use of bi-axial rotation therapy with cobalt^{60}, physical basis and application in the treatment of carcinoma of the cervix. *J. Canad. A. Radiologists*, Sept., 1959, *10*, 47–50. (From: Notre Dame Hospital, Montreal, Quebec, Canada.)

The author shows that eccentric arc therapy to each side of the pelvis produces isodose curves shaped to fit the pelvic anatomy. The degree of rotation varies from 150 to 210 degrees to each side. Radium therapy complements a high tissue dose of 6,000 r in six weeks to a large central pelvic volume. Ports 6×15 cm. are used. Radium delivers an additional dose of 2–3,000 mg. hr.

A very ingenious method of taking the contour of a prone or supine patient is described. It utilizes a magnetized metal tape inside a rigid ovoid device suggested by the Germans. The metal rods shape the metal strip to the correct contour. Dosimetry was by the Kodak type M film technique, read in a Welch densitometer. Forty-six patients have been treated. —*Jesshill Love, M.D.*

Friedman, Milton. Supervoltage (2-mvp) rotation irradiation of cancer of the bladder. *Radiology*, Aug., 1959, *73*, 191–208. (Address: 1016 Fifth Ave., New York 28, N. Y.)

This is a preliminary report on 81 patients with cancer of the urinary bladder treated with 2 mev. roentgen rays by the rotation technique. In most instances the lesions were advanced and the bladders were scarred by previous fulgurations and segmental resections. About three-fourths of the patients had been subjected to numerous surgical procedures of different types for successive lesions. A 2 mev. resonant transformer roentgen-ray generator was employed at a target-axis distance of 125 cm. The dose rate at the axis of rotation was 57 rads per minute. The patient was standing erect on a rotating platform during treatment. The technique of beam aiming utilized the right angle coordinate system with the aid of the Dresner pointer. In most instances the field size at the axis of rotation was 8×8 cm.

The maximum dose required to destroy cancer of the bladder was found to range from 6,000 rads in forty days to 11,000 rads in fifty-five days. Most doses for early papillary cancers ranged from 8,000 to 9,000 rads. These large doses can be given only with a well collimated beam of supervoltage roentgen or gamma rays, and with precision beam aiming.

Results from radical surgery, supervoltage rotation therapy, and a central intracavitary source are compared. Irradiation with the 2 mev. roentgen rays by the rotation technique achieved a 22 per cent three year arrest rate and 12 per cent five year arrest rate. The central intracavitary source yielded far better results, with a 56 per cent five year arrest rate. The five year survival following cystectomy as reported by Whitmore was 18 per cent. Radiation cystitis was more severe following supervoltage rotation therapy than following the use of a central intracavitary source.

In order to improve the results of irradiation of bladder cancer, the prevalent practice of multiple surgical procedures prior to irradiation must be discontinued and irradiation started when the disease is in an earlier phase.—*Arno W. Sommer, M.D.*

Rubin, Philip, and Buran, Robert. Supervoltage irradiation in bladder carcinoma: 1952–1958. *Radiology*, Aug., 1959, *73*, 209–225. (Address: P. Rubin, 266 Crittenden Blvd., Rochester 20, N. Y.)

Carcinoma of the bladder represents a group of tumors ranging from benign papillary carcinoma to a highly invasive anaplastic tumor. The variability of its course and lack of a generally acceptable classifi-

cation has obscured the problem. Recently proposed classification systems have emphasized two essentials known to influence prognosis: (1) the physical extent or depth of infiltration of the tumor; and (2) its inherent aggressiveness or histologic grade. Secondary factors influencing prognosis are the general state of health, associated urologic complications, recurrent lesions, previous surgery and previous radiation therapy.

The present report is based on a series of 78 cases seen at Strong Memorial Hospital in a six year period. Sixty-one of these were treated by 1 mev. rotational supervoltage irradiation. On cystoscopic examination before treatment, 63 per cent of the patients had multiple lesions. The most frequent anatomic sites of origin were the trigone, the bladder neck, and the lateral walls.

The daily tumor dose was 200 r, measured at the axis of rotation. The total tumor dose ranged from 5,000 to 6,000 r in five or six weeks, with doses of 3,000 r to 4,000 r reserved for palliative rather than curative purposes. In recent years 6,000 r delivered in six weeks has been considered the minimal tumoricidal dose. The field size usually ranged from 8×8 to 10×10 cm. Either 270° oscillation or 360° rotational therapy was employed. Radiation cystitis was the most common untoward reaction, occurring in 55 per cent of the cases. Ureteral obstruction, perforation or fistulas are infrequent complications during therapy. Persistent cystitis, calculus formation and hemorrhage are possible late complications.

For superficial, low grade carcinoma (Group I) the initial treatment of choice is a conservative surgical measure such as fulguration, transurethral resection of the tumor, or segmental resection. Supervoltage therapy should be used only as an alternative to total cystectomy and not as a prophylactic measure.

For deep, high grade carcinoma (Group II) supervoltage irradiation in curative dosage levels has the most to offer. Results suggest that cure rates are at least comparable and perhaps better than those obtained by radical surgical procedures, with less morbidity and mortality.

For metastatic stage, high grade carcinoma (Group III) supervoltage irradiation is effective to control pain and hematuria and is more easily tolerated than orthovoltage therapy. In selected cases, an ileal bladder may make it possible to deliver a curative, rather than a merely palliative, dose of irradiation.—*Arno W. Sommer, M.D.*

Markus, Benno. Oberflächenschichtbestrahlungen mit schnellen Elektronen eines Betatrons. (Irradiation of superficial layers with fast electrons from a betatron.) *Strahlentherapie*, Oct., 1959, *110*, 260–265. (From: Universitäts-Hautklinik, Abteilung Elektronenschleuder, Göttingen, Germany.)

The ideal distribution of energy in radiation therapy is a homogeneous dose of 100 per cent to a box-shaped layer of tissue and no irradiation to surrounding tissue, including deeper tissues. This can be attained only in grossest approximation by roentgen rays or any other quantum radiation. However, the 15 mev. Siemens betatron employed by the author with a 3 mev. electron beam, partially absorbed by thin plexiglass sheets placed in the radiation cone, yielded practically this ideal distribution of energy for tissue depths of 0.2, 0.4, and 0.6 cm.; *i.e.*, a flat, box-like layer of tissue to these depths received a 100 per cent dose and surrounding (including deeper) tissues received much smaller doses. (Measurements were made in a water-equivalent phantom.)—*Henry G. Moehring, M.D.*

RADIOISOTOPES

Mack, Robert E., and Ortmeyer, Leroy F. Radioiodine in the evaluation of the thyroid nodule. *Surg., Gynec. & Obst.*, Dec., 1959, *109*, 668–672. (From: St. Louis Veterans Administration Hospital and Department of Internal Medicine, St. Louis University School of Medicine, St. Louis, Mo.)

It is well known that certain diseases of the thyroid gland cause a difference in the ability of the gland or parts of the gland to concentrate I^{131}. The scintigram produced by an automatic scanning counter has given considerable help in the evaluation of thyroid disease and especially of nodules in the thyroid gland. In the present report, the size of the nodules was an important factor in that nodules under 1.5 cm. in size were not well demonstrated if the concentrating ability was poor.

The authors used plastic thyroid models filled with varying concentrations of I^{131} solutions and with known defects in the models simulating nodules in order to evaluate detection of the defects using improvements in the instruments and a three dimensional technique. An automatic scanning counter with a single channel pulse height analyzer to discriminate against all radiation not directly emanating from the source was used. A focusing type lead shield was also used. The output of the scaling circuit of the scanner was connected to a count rate meter and its graphic recorder. Any variations in the concentration of I^{131} were indicated on the scintigram and recorded as a parabolic curve on the counting rate record. Three dimensional models were constructed from each scintigram by transferring the counting rate meter record to cardboards and mounting them in a slotted wooden block.

The study showed little improvement in the ability of the modified counting system to detect small areas of poor I^{131} concentration. The three dimensional model constructed from the scintigram was more accurate in demonstrating abnormalities than was

simple inspection of the scintigram.—*Charles Cooley, M.D.*

Goolden, A. W. G. Effect of thyrotropic hormone on the accumulation of radioactive iodine in thyrotoxicosis. *J. Clin. Endocrinol. & Metabol.*, Oct., 1959, *19*, 1252–1257. (From: Medical Research Council Radiotherapeutic Research Unit, Hammersmith Hospital, London, W. 12, England.)

The investigation reported by the author was designed to study the effect of the thyroid stimulating hormone on the accumulation of iodine in the thyroid gland in thyrotoxicosis. Twelve thyrotoxic patients with diffuse or nodular goiter were chosen for study. Six had received no treatment. The thyroid clearance rate was used to detect any thyroid change.

$$\text{Thyroid clearance} = \frac{\text{Rate of change of thyroid } I^{131}}{\text{Plasma concentration } I^{131}}.$$

In every case the thyroid clearance was increased after administration of the thyroid stimulating hormone and the amount of I^{131} excreted was less. The author states that most cases showed an increase in uptake at one, two, four, and six hours but no increase was apparent at twenty-four hours. The degree of thyrotoxicosis had little bearing on the thyroid clearance. It seems as though the pituitary-thyroid relationship does not function normally in thyrotoxicosis. The data show that the thyroid responds to thyrotropic stimulation in the thyrotoxic patient.—*Jesshill Love, M.D.*

Kammer, Huldrick, and Goodman, Morton J. Sterility after radioiodine therapy for metastatic thyroid carcinoma. *J.A.M.A.*, Dec. 5, 1959, *171*, 1963–1965. (From: Department of Medicine, University of Oregon Medical School, Portland, Ore.)

A twenty-four year old man was found to have diffuse nodular densities throughout both lung fields on a routine chest roentgenogram in 1952. Physical examination showed an enlarged left lobe of the thyroid gland and a small lymph node in the left side of the neck. The lymph node was found to contain metastases from a carcinoma of the thyroid gland. A thyroidectomy and radical left neck dissection were done and the primary lesion was found to be a mixed adenocarcinoma of the thyroid with a predominantly papillary pattern. The lung lesions were concluded to represent metastases. The patient was treated with 246 mc I^{131} in July, 1952 and in February, 1953 an additional dose of 317 mc I^{131} was given. Myxedema developed in July, 1953 and he was placed on triiodothyronine for replacement therapy; he has continued on it since that time. The patient has remained well except for some respiratory limitation due to widespread pulmonary fibrosis.

In 1956, fertility studies showed absence of sperm in the ejaculate and testicular biopsies revealed a complete lack of germ cells in the tubules and no evidence of spermatogenesis. Moderate fibrosis of the tunica propria was present but the basement membrane showed little change. The seminiferous tubules were reduced in size. The Sertoli and Leydig cells were normal. The urine showed an increased amount of pituitary gonadotropins with the titer being equivalent to that of a castrated man.

One other case was reported by Duffy of a fifty-year old man in whom metastatic thyroid carcinoma had been treated with I^{131}. Testicular biopsy revealed atrophic changes and absence of spermatogenesis. Animal experiments have shown that the testes are quite vulnerable to internally administered radioactive isotopes and that rapid and complete recovery takes place if the injury is minimal.

Further studies of similarly treated patients may reveal more examples of radiation injury to the male gonads.—*Charles Cooley, M.D.*

Paris, Jaime, McConahey, William M., Owen, Charles A., Jr., Woolner, Lewis B., and Bahn, Robert C. Iodide goiter. *J. Clin. Endocrinol. & Metabol.*, Jan., 1960, *20*, 57–67. (From: Sections of Medicine, Clinical Pathology, Surgical Pathology, and Pathologic Anatomy, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

The development of a goiter, with or without myxedema, is a very unusual complication of the protracted use of iodides. Less than a score of such instances have been reported in the English literature notwithstanding the large number of patients who take iodides for asthma and other diseases.

In the present paper 5 cases of iodide goiter are reported all of which showed parenchymatous hypertrophy of the thyroid on biopsy. Intake of iodides for uninterrupted and prolonged periods ranged from thirteen months to more than seven years. Tracer studies with I^{131} were performed on all of these patients at intervals of six and twenty-four hours after the administration of the I^{131}. The uptake by the thyroid gland, when enough time had elapsed after the last dose of iodide-containing medicaments, was uniformly increased. In the majority of instances, it was impossible to interrupt the administration of iodide-containing medicaments, but the goiter and the manifestations of myxedema disappeared during treatment with desiccated thyroid. In 1 case, in which use of iodides was permanently discontinued and treatment with thyroid was not instituted, complete resolution of the myxedema and the goiter was seen within three months. In 1 other case, the preoperative diagnosis was Hashimoto's thyroiditis, and to the surprise of both surgeon and clinician the histologic appearance of the thyroid was that of parenchymatous hypertrophy.—*D. S. Kellogg, M.D.*

GROOS, EKKEHARD. Untersuchungen von Futter und Milch auf ihren Gehalt an Strontium 90. (Investigations of the strontium 90 content of feed and milk.) *Strahlentherapie*, Sept., 1959, *110*, 66–69. (From: Max-Planck-Institut für Biophysik, Frankfurt/Main, Germany.)

Between January and May, 1958, successive milk samples from a cow were assayed for strontium 90 content. Though the animal was being fed from the same supply of stored feed (hay and ensilage), the milk showed a maximum strontium 90 content in February; this finding is unexplained. When fresh grass was fed, the strontium 90 content of the milk rose abruptly. This was anticipated because of the high strontium 90 content of the fresh grass. (Samples of the grass also had a considerable content of strontium 89).—*Henry G. Moehring, M.D.*

MESCHAN, I., QUINN, JAMES L., WITCOFSKI, RICHARD L., and HOSICK, THOMAS A. The utilization of radioactive zinc and manganese in an effort to visualize the pancreas. *Radiology*, July, 1959, *73*, 62–70. (Address: I. Meschan, Bowman Gray School of Medicine, Winston-Salem, N. C.)

A number of investigators have demonstrated that pancreatic tissue has a selective affinity for certain substances such as zinc and manganese compounds. Thus far, no useful nonsurgical means for pancreatic visualization have been found. A brief historical review is presented to support this contention. The object of the present study was to determine: (1) the fate of zinc and manganese compounds in relation to the liver, pancreas, kidney and spleen in dogs, using radioactive isotopes of these elements; (2) whether radioactive scan studies would be helpful in delineating the liver and pancreas in dogs; and (3) whether characteristic uptake curves for these elements could be obtained over the pancreas. The following compounds were used: (1) manganese chloride (Mn^{54} and Mn^{52}), (2) zinc chloride (Zn^{65}), (3) zinc sulfadiazine (Zn^{65}), and (4) zinc complex of 3′-6′-aminofluorescein.

External uptake measurements over the pancreas were made at intervals of eight to twenty-four hours in 15 dogs following the administration of 100 μc of Zn^{65} chloride and in 2 additional dogs receiving Zn^{65} sulfadiazine and Zn^{65} complex of 3′-6′-aminofluorescein. The animals were sacrificed at different time intervals for bioassay of the liver, kidney, spleen and pancreas. The radioactivity measurements in terms of concentration per gram weight of organ were correlated with the uptake curves obtained over the pancreas just prior to the time of sacrifice. The same procedure was used on 10 other dogs receiving Mn^{54} and Mn^{52}.

Graphs are presented to show the percentages of the various compounds which were recovered from the different organs at different time intervals, and these are compared with graphs showing uptake curves over the pancreatic areas of the dogs before sacrifice. A biphasic uptake curve was obtained representing organ concentration for both zinc and manganese in the liver and pancreas. Thus far, however, this curve has not proved to be clinically useful. Concentration of Zn^{65} in the pancreas was found to be greater than that of manganese. The attempted scanograph studies for delineation of the pancreas were found to be insufficiently detailed to be applicable either clinically or experimentally.

The authors offer a number of suggestions concerning the possible future application of their data to human study.—*Z. Petrany, M.D.*

FORREST, A. P. M., BLAIR, D. W., BROWN, D. A. PEEBLES, STEWART, HELEN J., SANDISON, A. T., HARRINGTON, R. W., VALENTINE, J. M., and CARTER, P. T. Radio-active implantation of the pituitary. *Brit. J. Surg.*, July, 1959, *47*, 61–70. (From: University Department of Surgery, Pathology and Ophthalmology, Western Infirmary, Glasgow; Department of Physics, Western Regional Hospital Board; Department of Metallurgy, Royal College of Science and Technology, Glasgow, Scotland.)

During a period of four years the authors treated 141 patients, most of whom had disseminated carcinomatosis, by radiation pituitary ablation techniques. Forty-five patients were treated by the implantation of 10 to 20 mc of radon into the pituitary fossa. In the first 25 patients, two to four 5 mc seeds were inserted into the sella turcica, one through each nostril, and a single 8 mc seed was introduced into each half of the fossa. In 8 patients so treated there was progressive loss of vision, developing up to twelve months after implantation. Rhinorrhea occurred in 2 patients, with fatal meningitis developing in 1. Pituitary destruction was estimated in 20 cases brought to autopsy to range between 10 and 90 per cent. Satisfactory regression of disseminated breast disease occurred in 11 of 36 patients treated for the control of this process.

Fifty-four patients were treated for disseminated carcinomatosis from primary breast tumors with one exception, by transnasal sellar implantation of two rods of irradiated yttrium oxide containing a total of 8–17 mc of Y^{90}. Serious visual loss was encountered in only 1 patient, but there were 10 instances of postoperative rhinorrhea. In 23 patients brought to postmortem examination, 90 per cent destruction of the pituitary gland was found in 15 cases. Clinical results in this group of patients were compared with those observed in an alternating series of patients treated by adrenalectomy and oophorectomy, and were found to be roughly the same. Results were not

promising in a small group treated for recurrence of the disease following adrenalectomy and oophorectomy.

A second method of Y^{90} implantation was devised, in which two yttrium sources, totaling between 9 and 14.5 mc of Y^{90}, were inserted into the sella turcica within threaded screws which were allowed to remain in place in the bone of the anterior sellar wall. Of 42 patients treated with the new technique, visual acuity was impaired in all but 2. Rhinorrhea was encountered in 3 others. Of 12 pituitary glands treated in this way which were examined at autopsy, virtually complete destruction was seen in each. Early clinical results in this group are promising.

The production of complications is analyzed in detail. The much higher rate of occurrence of serious visual defects is attributed to gamma radiation in the radon treated cases. Y^{90} emits a beta particle (peak energy = 2.24 mev.) and, in the few instances in which optic nerve damage did occur, it was less extensive in nature and appeared to be produced by improper positioning of one or more of the rods high in the sella. Rhinorrhea was thought to be due principally to radionecrosis of the diaphragm of the sella, occurring when one of the radiation sources lay in contact with, or in proximity to, this structure.

It would appear that implantation of small radioactive, beta emitting sources in the sella is an effective method of producing hypophysectomy in the management of disseminated neoplastic disease. The technique of implanting sources contained within screws which are left in place in the sella is an ingenious one and worthy of further study.—*F. J. Bonte, M.D.*

Dogliotti, A. M., Ruffo, A., and Molinatti, G. M. Implantation of the pituitary with yttrium^{90} in five cases of acromegaly. *Panminerva med.*, May, 1959, *1*, 15–18. (Address: A. M. Dogliotti, Clinica Chirurgica, Corso Polonia 14, Torino, Italy.)

The authors have undertaken treatment of 5 cases of acromegaly using pituitary implantation of four to five small sources of Y^{90} of about 5 to 6 mc strength. The sources were inserted through a cannula by the nasal trans-sphenoidal route described by many authors. It is pointed out that distant complications are minimized by using a pure beta emitter, such as Y^{90}. An immediate complication in this series, as in all others, is polyuria which disappeared within three months. Several cases showed reversal of a negative calcium balance. Clinical results were encouraging, although apparently none of the patients has been observed for more than twenty-two months after treatment.—*Frederick J. Bonte, M.D.*

CHEMOTHERAPY

Loken, Merle K., Kim, Young S., Mosser, Donn G., and Marvin, James F. The effect of combined irradiation and chemotherapy on cancer growth; with special reference to studies with folic acid analogues. *Radiology*, Aug., 1959, *73*, 166–174. (Address: M. K. Loken, University of Minnesota Hospitals, Minneapolis 14, Minn.)

"Therapeutic ratio," which has been defined as the ratio of the radiosensitivity of tumor tissue relative to surrounding normal tissues, might be increased through either a preferential protection of normal tissues or by selective sensitization of tumor tissue to the effects of ionizing radiations. The latter result may be achieved either through enhancement of the direct effect of radiation on cellular components or through inhibition of recovery processes. Synergistic action of certain chemicals and radiation in their effect on malignant tumors might be expected because of the evidence that alternate metabolic pathways exist in cells, so that blocking one path by administration of a particular chemical or by radiation therapy may not produce the desired response. However, if both a major pathway and its alternates are blocked, a specific result might be achieved, such as the inhibition of nucleic acid synthesis which is in some way dependent upon folic acid metabolism. Inasmuch as the folic acid antagonists and ionizing radiations have some similarities in their effects on biologic structures, a study of their possible synergistic action was made.

Mice with strain-specific transplantable mammary carcinoma were subjected to total body irradiation, both with and without previous intraperitoneal injection of various folic acid antagonists administered 30 minutes prior to the irradiation. Two exposure doses were used in this study, one of which was near the LD 50/30 and the other near the LD 100/30. Analysis of the data showed that each of the antagonists potentiated the lethal effects of roentgen rays, both in terms of mortality rate and per cent mortality. This indicated that there is no preferential protection of normal tissues; rather, the radiation effect is enhanced. From these results one is not encouraged to believe that the folic acid analogues could be of benefit in combined therapy. Only if they selectively sensitize tumor tissue to the effects of radiation would they be of any value.

In order to evaluate this further, additional experiments were performed in which similar mice were given local irradiation to the tumor both with and without previous intraperitoneal injection of one of the analogues. Controls were also included for each experiment. In the analysis of these results it became apparent that groups of mice receiving the same treatment in all of the experiments exhibited a similar response, although there were individual and tumor type variations. The tumors in the group of mice which received combined analogue and roentgen-ray treatment regressed faster, remained smaller for a longer period of time, and exhibited a rate of re-

growth which was less rapid than that for the tumors in any other group.

Whether or not the potentiating effect observed with the analogues in these studies may have been the result of interference with metabolic processes within the injured tissues or of blocking the synthesis of metabolites at other sites, these antimetabolites may be of definite value in combined therapy inasmuch as their radiation is normally localized to the area involved by the malignant process.

The approach to this study was somewhat empirical. Improvement in the experimental results of combined therapy may result from the study of other metabolic antagonists and combinations of antagonists, variations in dosage of the antagonist and radiation, time relationship between administration of the antagonist and radiation, effect of multiple treatments, and utilization of tumors other than mouse mammary carcinoma.—*Walter H. Jarvis, Jr., M.D.*

SOLAN, M. J. Prochlorperazine and irradiation sickness. *Brit. M. J.*, Nov. 21, 1959, *2*, 1068–1069. (From: St. Thomas's Hospital, London, England.)

The suppression of side effects of radiation therapy such as nausea, vomiting, lassitude and anorexia has been made easier in the past few years with the introduction of a number of anti-emetic drugs that have primary action on the vomiting center. Prochlorperazine was found by Ducrot and Koetschet in 1956 to be four times more effective than chlorpromazine and superior to any of the other drugs used in suppression of artificially induced vomiting in experimental animals.

The author made observations on 57 patients who were expected to suffer side effects from therapeutic irradiation. Twelve cases did not develop symptoms. The remaining 45 cases were given either 30 mg. of prochlorperazine per day, 60 mg. of pyridoxine (vitamin B_6) or placebo tablets for their symptoms. Prochlorperazine was found to be more effective in relieving the symptoms than pyridoxine or the inert tablets. No significant side effects such as marked drowsiness were encountered and no toxic effects were found. The maximum benefit from prochlorperazine was obtained when it was given with the first exposure to irradiation or as soon as possible after the onset of symptoms.—*Charles Cooley, M.D.*

D'ANGIO, GIULIO J., FARBER, SIDNEY, and MADDOCK, CHARLOTTE L. Potentiation of x-ray effects by actinomycin D. *Radiology*, Aug., 1959, *73*, 175–177. (Address: G. J. D'Angio, The Children's Medical Center, 300 Longwood Ave., Boston 15, Mass.)

There have been previous reports that roentgen-ray effects on normal tissues have been enhanced by combining roentgen irradiation with the intravenous administration of actinomycin D, with encouraging responses to this form of combined therapy in a variety of neoplasms. In this study of 126 patients who received 220 courses of this form of combined treatment, it has been noted that the reaction in normal structures occurs after smaller doses of radiation and healing proceeds at a more rapid rate than following roentgen therapy alone. Actinomycin D alone can reactivate latent radiation effects in tissues previously irradiated but having since returned to normal in appearance. This response in the skin is sharply restricted to the previously irradiated areas and is identical in type to that originally produced by the roentgen irradiation, being most accentuated when only a brief interval separates the course of radiotherapy and chemotherapy. These enhanced effects can lead to dangerously severe local reactions if high doses of each agent are used together or successively, or if a patient proves to be especially sensitive to the combination. Experimental studies of the effects of combined treatment on the skin of normal mice confirmed the clinical observations.

Although an augmented effect on normal tissues by combined treatment has been established, proof of a similar response in neoplastic tissues is more difficult to evaluate. However, it was noted that some neoplasms, such as the malignant melanoma, usually considered to be radioresistant, have responded well to combined therapy in some individuals. The most favorable responses have been observed in patients with Wilms's tumor, Ewing's tumor, hemangioendotheliosarcoma, and rhabdomyosarcoma. The combined treatment in low dosage appears to be as effective as higher doses of either modality alone, and there is some evidence of a longer-lasting beneficial effect. Potentiation of roentgen-ray effects in normal tissues has not limited the total amount of roentgen therapy that can be given to any patient; however, interruptions to allow recovery have not been infrequent. The effects in some tumors have been striking without apparent enhanced reactions in the normal skin or mucous membranes, and, conversely, the augmentation of reaction in normal structures has not always been accompanied by similar potentiation in the underlying neoplasm.—*Walter H. Jarvis, Jr., M.D.*

KURNICK, N. B., FEBER, BERNARD H., MONTANO, ANDREW, GERDES, JAMES C., and NAKAMURA, ROBERT. Some observations on the treatment of postirradiation hematopoetic depression in man by the infusion of stored autogenous bone marrow. *Ann. Int. Med.*, Dec., 1959, *51*, 1204–1219. (Address: N. B. Kurnick, Veterans Administration Hospital, Long Beach 4, Calif.)

Donation of bone marrow in man has had merely transitory success lasting only a few weeks. Multiple blood group compatibility is no assurance of donor

tissue compatibility. Roentgen-ray suppression of the immune response mechanism requires high dosage which damages many tissues. Furthermore, in successful transplants, homologous disease ensues due to antibody production by donated leukocytes against recipient tissues.

In the present study, these difficulties were overcome by transfusing the patient with his own (autologous) stored bone marrow. The bone marrow is collected from the patient's both iliac bones prior to radiation therapy using siliconized apparatus containing 2–5 units of heparin diluted in 30 per cent glycerol and frozen at the rate of 1° C. per minute to minus 79° C. It is then stored.

It was discovered that regeneration and hyperplasia of severely radiation-depressed marrow occurs in approximately one and one half to four weeks following infusion of stored autologous bone marrow. The bone marrow aspirates in control subjects taken three months after intensive radiation therapy were markedly hypoplastic and ten months later were still moderately depressed, in contrast to the hyperplasia of marrow in the patients infused within one and one half to four weeks.

Bone marrow is now routinely collected from all patients with normal marrow who are about to receive suppressive bone marrow irradiation or chemotherapy for malignancies. Intensive therapy is then given in a short period of time with less concern for the leukopenia and thrombocytopenia that results. As an adjunctive therapy, restored bone marrow is then reinfused.—*Bernard Loitman, M.D.*

Glicksman, Arvin S., Rawson, Rulon W., and Nickson, James J. Modification of late radiation injury with L-triiodothyronine. *Radiology*, Aug., 1959, *73*, 178–190. (Address: A. S. Glicksman, Memorial Center, 444 East 69th St., New York 21, N. Y.)

Severe changes due to the late effect of exposure to ionizing radiation are seen periodically. These changes characteristically have shown no response to many different types of treatment. Observation that the production of hyperthyroidism seemed to exert a beneficial effect on severe post-irradiation changes in a group of patients with carcinoma of the thyroid led to the present study.

Sixty-nine patients have been treated for sufficiently long periods to permit evaluation of the effect of 3,5,3′-L-triiodothyronine (T-3) on established postirradiation changes. Well over half of the patients with damage which had existed from two to thirty years showed good to excellent response to this T-3 therapy. This response is thought to be due to alteration of the metabolism of the damaged tissue. In many instances, long standing ulcers completely healed and subcutaneous fibrosis decreased.

The authors feel that if these damage patterns can be modified by such treatment the applicability of ionizing radiation to human disease will be broadened. An effective therapy for radiation damage could either decrease damage to the normal tissue, if the current time-dosage factors are preserved, or extend the probability of a cure by permitting the use of larger time-dosage relationships for the same degree of normal tissue damage.

Six case reports are presented illustrating the results of treatment.—*D. N. Dysart, M.D.*

MISCELLANEOUS

Wideröe, Rolf. Integraldosen für 200 keV-Röntgen- und für Megavoltstrahlen. (Integral doses for 200 kv. roentgen rays and for megavolt radiation.) *Strahlentherapie*, Sept., 1959, *110*, 1–9. (From: Radiotherapeutische Klinik der Universität Zürich, Zürich, Switzerland.)

This work concerns itself only with the integral dose from radiation. All considerations of distribution of depth dose, skin dose, exit dose, differential absorption in various tissues, scattering of radiation, and relative biologic effect are specifically excluded.

Conclusions are based on data from radiologic literature and from the author's own measurements. The most satisfactory depth doses per unit of integral dose come from the use of optimum energy electron beams. This investigation "shows plainly that the future belongs to therapy with an electron beam." For all practical purposes, the electron beam yielded the lowest specific integral dose.

In comparing 200 kv. roentgen rays with cobalt 60 radiation at a depth of 10 cm. in 20 cm. of body thickness, the specific integral dose of the 200 kv. beam was 2.35 times greater than of the cobalt 60 beam.—*Henry G. Moehring, M.D.*

Frantz, F. S., Jr., and Wyckoff, H. O. Attenuation of scattered cesium-137 gamma rays. *Radiology*, Aug., 1959, *73*, 263–266. (From: National Bureau of Standards, Washington, D. C.)

Protection of personnel from scattered irradiation necessitates the construction of adequate protective barriers. Lead and concrete are the most commonly used materials in such barriers. Attenuation curves in these substances of the secondary radiations from various sources would provide useful data for the effective and economic construction of safe protective barriers.

Attenuation data for scattered gamma rays have been obtained in two ways: (1) using a phantom to duplicate the conditions under which a patient scatters the incident beam; and (2) measuring the radiation scattered at various angles from a concrete wall at fixed distances from the source. The present study uses the latter method with Cs^{137} as the radiant gamma source. The geometric arrangement of the

experimental apparatus is shown. Data for attenuation of the secondary irradiation in lead and concrete for various scattering angles are presented and compared with theoretic curves based on Compton single scattering and also with curves obtained using Co^{60} as a source. The relationship of scattering angle and intensity of scattered radiation shows that, as the scattering angle increases, the scattered dose per unit of incident dose decreases in a linear manner. This is true for scattering angles from 0–90°, but beyond 90° the decrease becomes minimal.

As demonstrated in the present study, the attenuation of scattered Cs^{137} gamma rays of an energy computed by the Compton relation agrees with that obtained for scattered Co^{60} gamma rays of the same energy, with an accuracy sufficient for use in protection determinations.

The technical limitations of the experimental arrangement are pointed out and discussed.—*Z. Petrany, M.D.*

Stanton, Leonard. Penumbra measurements of supervoltage and cobalt-60 machines by a photographic method. *Radiology*, Aug., 1959, *73*, 253–262. (Address: Department of Radiology, Hahnemann Medical College and Hospital, Philadelphia 2, Pa.)

Cobalt 60 sources now in use range up to 2.54 cm. diameter and higher, so that penumbras of several centimeters are not uncommon, as compared with a few millimeters in 250 kv. therapy. Thus, the importance of a convenient method of measuring the cobalt penumbra, such as the one outlined by the author, is readily apparent. Although ionization chambers would have permitted the most accurate radiation dosage measurement, technical difficulties associated with their use (size, positioning, etc.) made the positioning of an 8×10 inch film along a millimeter scale much more feasible for the author's purposes.

The technical aspects of penumbra measurement, including type of film, densitometry using a "photovolt" transmission photomultiplier type densitometer, and the nature of penumbra formation, are discussed in detail, as well as the effect of source size and collimator design. The intensity variation in the penumbral region for various skin source distance and tumor source distance values are given for 4 types of cobalt units and 2 different supervoltage units. The Van de Graaff supervoltage unit exhibited the smallest penumbra while the small (hectocurie) cobalt units exhibited the largest penumbras, due basically to the necessity for short treatment distances resulting from the use of weaker sources of large diameter.—*John S. Alexander, M.D.*

Pohlit, Wolfgang. Zur Frage der bei Betatronbestrahlung im Phantom auftretenden Neutronenverteilung. (The distribution of neutrons arising in a phantom irradiated by a betatron.) *Strahlentherapie*, Oct., 1959, *110*, 240–247. (From: Max-Planck-Institut für Biophysik, Frankfurt/Main, Germany.)

The distribution of neutrons arising in water and paraffin phantoms irradiated by a 35 mev. betatron was studied by means of indium probes. The neutrons entering the phantom came from heavy atoms (lead) and fell off exponentially with a half value layer of 5.8 cm. in paraffin. The neutrons from the reaction $C^{12}(\gamma, n)C^{11}$ increased in number at greater depth in the phantom to reach their maximum at about 10 cm. in paraffin and then decreased slowly—with a half value layer of about 50 cm. in paraffin. At 50 rad/min. and a distance of one meter, each of these two groups of neutrons was of the magnitude of 10^4 n/cm.^{2} sec.—*Henry G. Moehring, M.D.*

Harder, Dietrich, Kretschko, Julius, and Pohlit, Wolfgang. Die Messung der bei Betatronbestrahlung erzeugten Luftaktivität. (The measurement of radioactivity produced in air by betatron radiation.) *Strahlentherapie*, Oct., 1959, *110*, 234–239. (From: Max-Planck-Institut für Biophysik, Frankfurt/Main, Germany.)

At a dose output of 50 rad per minute at a one meter target distance, ionization chamber measurements show that the oxygen in the air of a betatron treatment room becomes radioactive to an intensity of 3×10^{-11} curie/cm.^{3}; this is seven times the "permissible" degree of radioactivity. The nitrogen of the air becomes radioactive to an intensity of 1.5×10^{-12} curie/cm.^{3}; about one fourth of the "permissible" degree.

For the patient, these degrees of radioactivity of the air spell only about one thousandth of the tumor dose, but the important consideration is that air from the treatment room does not get into the control room.—*Henry G. Moehring, M.D.*